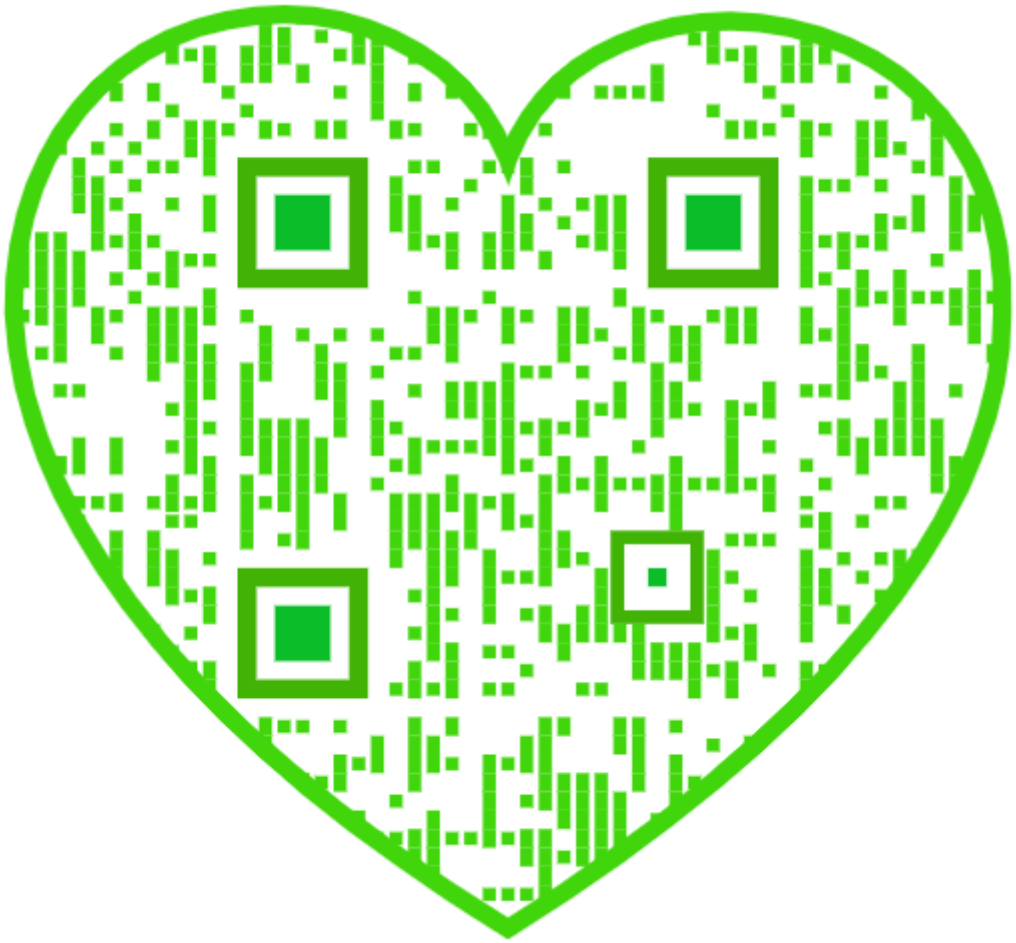

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PRACTICAL MEDICINE.

BY

F. MORTIMER LAWRENCE, A. M., M. D.,

Assistant in Practice of Medicine, Hahnemann Medical College;
Chief of Medical Clinic, Hahnemann Hospital Dispensary,
Philadelphia.

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TO

MY MOTHER.

PREFACE.

This book is intended for students, not advanced workers. The endeavor has been to set forth concisely those fundamental facts which are requisite to the successful practice of medicine. Pathological processes rather than the details of morbid anatomy have been described, with the object of co-relating the symptoms of disease to the underlying changes. In connection with diagnosis the more important modern laboratory methods have been included; and in addition each section is preceded by a brief resume of the essential points to be ascertained by interrogation of the patient, and of the physical methods by which the examination should be completed.

The work as a whole is based upon the masterly teachings of Goodno, and to him in particular must be attributed the practical, though necessarily condensed, directions as to treatment. In the preparation of the articles dealing with semi-surgical conditions, notably appendicitis, intestinal obstruction, cholelithiasis, and nephrolithiasis, the author has profited by the advice and kindly criticism of Dr. William B. Van Lennep. The sections devoted to clinical methods and neurology represent the teachings of Dr. Clarence Bartlett, while to Dr. Weston D. Bayley the author is indebted for a careful review of that portion of the book dealing with the clinical examination of the nervous system. Constant reference has been made to the writings of other authorities, however, and it is hoped that the work will afford a clear perspective of the modern practice of medicine.

FREDERIC MORTIMER LAWRENCE.

OCTOBER 17, 1901.

BIBLIOGRAPHY.

The author acknowledges particular indebtedness to the following works and their respective writers:

Allbutt, T. Clifford: A System of Medicine, 1896.
Broadbent, William H.: Heart Disease, 1900.
Bury, Judson S.: Clinical Medicine, 1894.
Butler, G. R.: Diagnostics of Internal Medicine, 1901.
Cabot, Richard C.: Examination of the Blood, 1901.
Cabot, Richard C : Physical Diagnosis, 1900.
Church, A. and Peterson, F.: Nervous and Mental Diseases, 1900.
Coles, A. C.: Diseases of the Blood, 1898.
Finlayson, James: Clinical Manual, 1891.
Fowler, J. K., and Godlee, R. J.: Diseases of the Lungs and Pleuræ, 1898.
Goodno, William G.: Practice of Medicine, 1896.
Herter, Christian: Diagnosis of Diseases of the Nervous System, 1892.
Hutchison and Rainy: Clinical Methods, 1897.
Hughes, Richard: Manual of Pharmacodynamics, 1886.
Klemperer, G.: Clinical Diagnosis, 1898.
Musser, John H.; Medical Diagnosis, 1900.
Osler, William: Practice of Medicine, 1901.
Purdy, Charles: Practical Urinalysis and Urinary Diseases, 1900.
Raue, C. G.: Special Pathology and Therapeutics, 1895.
Raue, C. S.: Diseases of Children, 1899.
Reference Handbook of the Medical Sciences, 1901.
Strümpell, A.: Practice of Medicine, 1901.
Thompson, H. Campbell: Nervous Diseases, 1899.
Twentieth Century Practice of Medicine, 1900.
Tyson, James: Practice of Medicine, 1901.

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INFECTIOUS DISEASES.

An infectious disease is one caused by the invasion and reproduction within the body of pathogenic micro-organisms.

Contagion, the direct communication of disease from one person to another, occurs in those infectious diseases in which the virus is discharged from the body by channels which render its transportation to another easy. For example, in scarlatina, measles and smallpox the virus is eliminated by the skin, tiny scales of which are easily carried by the air. On the other hand, in malaria the virus is not discharged at all, and contagion is unknown; while in typhoid it is eliminated only in the urine and feces, and ordinary cleanliness renders contagion impossible.

Infection is conveyed by various media, including:

1. *Fomites*, *i. e.*, substances, such as clothing, furniture and domestic animals, which have been in direct contact with the infected patient. Smallpox and diphtheria in particular are apt to be conveyed in this manner.

2. *Water.*—This may become contaminated with the discharges of infected patients. Typhoid and cholera are among the diseases thus conveyed.

3. *Milk.*—This may convey contagion from a tuberculous cow, but more often it is contaminated by water used to wash the milk cans. Typhoid epidemics have originated in this way.

4. *Dust.*—Particles of dust frequently carry with them micro-organisms. In particular, the sputum of tuberculous patients, drying in the air, is caught up with the dust and the bacteria enter the lungs with the inspired air.

5. *Insects*, etc.—Typhoid, cholera, etc., may be spread by flies, which after contact with the infected excreta, carry the virus to deposit it on food; malaria is conveyed by mosquitoes, and bubonic plague by rats.

6. *Atmospheric air* was formerly regarded as a carrier of infection, but it is probable that the responsibility rests with dust, insects, etc., rather than with the air itself.

Infective agents gain access to the body through the respiratory system, through the intestinal system, or through some break in the skin. At the point of invasion the micro-organisms are met by resistance on the part of the organism (phagocytosis?) and are either destroyed or, having gained a foothold, proceed to a rapid reproduction; while coincidently with their rapid increase in numbers they elaborate certain chemical products which, disseminated through the body, act as violent toxins. In case the body can overcome the invaders at the outset, the individual is said to be *immune;* if, however, the infective agents are successful in overcoming the resistance of the body, the individual is said to be *susceptible.*

Immunity, the resistance of the living organism to infection, varies greatly in different individuals. It occurs in two forms: *natural immunity*, the natural and constant resistance of a healthy animal to certain diseases; and *acquired immunity*, the resistance resulting from accidental circumstances such as a previous attack of the same disease (yellow fever, etc.). In addition an *experimental immunity* may be induced by various laboratory manipulations.

As a corollary to these facts, it follows that **susceptibility** to infection varies widely in different animals, in different races, in different men, and in the same man at different times. In man this susceptibility is influenced by—

1. *Race.*—For example, the negro races resist infection with

malaria or yellow fever, but are particularly susceptible to smallpox and tuberculosis.

2. *Heredity.*—A family predisposition to certain fevers is common. Cancer and tuberculosis are regarded as diseases more or less hereditary. It is necessary, however, to consider the possible greater exposure to infection in these cases as, for instance, in the case of a child with a tuberculous parent.

3. *Environment.*—The influence of certain seasons, exposure to extremes of temperature, fatigue, and starvation tend to abolish resistance to micro-organic invasion in individuals not ordinarily susceptible.

4. *Pre-existing Diseases.*—Certain pre-existing lesions afford ready ingress and suitable environment for pathogenic micro-organisms. For instance, diabetic patients are particularly susceptible to the tubercle bacillus.

5. *Dosage of Bacteria.* The severity of an infection is, to a certain degree, in direct proportion to the amount of the virus.

The aim of modern investigators is to discover the means by which nature induces immunity in the individual, and to apply this knowledge toward the prevention and cure of disease. Their results thus far are embodied in the various therapeutic serums.

PERIODS OF INCUBATION AND INFECTIOUSNESS.

The time elapsing between the invasion of the organism and the development of the symptoms is known as the *period of incubation.* It varies considerably in the same disease according to the virulence of the infection and the susceptibility of the patient. The *period of infectiousness* represents the time during which the individual is liable to communicate the disease to others, and during which, in consequence, isolation and other precautions should be continued.

	INCUBATION.	INFECTIOUSNESS.
Typhoid,	7–22 days.	Onset to end of second week of convalesecnce.
Typhus,	5–21 days.	Onset to end of three weeks.
Relapsing,	5–16 days.	Onset to end of one week.
Dengue,	3–5 days.	Onset to end of one week.
Influenza,	1–5 days.	Onset to end of two weeks.
Yellow Fever,	1–14 days.	Onset to end of one week.
Malaria,	6–21 days.	Onset to disappearance of plasmodium.
Asiatic Cholera,	1–7 days.	Onset to end of one week.
Diphtheria,	2–7 days.	Incubation to three weeks or more after convalescence.
Scarlatina,	2–10 days.	Onset to end of desquamation.
Measles,	7–14 days.	Earliest prodromes to three weeks after recovery.
Rubella,	5–21 days.	Three days before rash to end of desquamation.
Small-pox,	8–15 days.	Onset to end of desquamation.
Chicken-pox,	12–19 days.	Onset to end of desquamation.
Whooping Cough,	7–14 days.	Onset to end of whooping period.
Mumps,	14–25 days.	Onset to end of three weeks.
Bubonic Plague,	3–6 days.	Onset to disappearance of symptoms.

In many of these diseases, particularly smallpox and diphtheria, conveyance of infection by fomites is possible for periods of months and even years. Only by thorough disinfection can this be prevented.

THE CLINICAL ASPECTS OF INFECTIOUS DISEASE.

The clinical manifestations attending infectious disease are, in general, those indicated by the term *fever*. They include elevation of temperature (*pyrexia*), acceleration of pulse and respiration, disturbances of the gastrointestinal and nervous systems (nausea, vomiting; pain, delirium), and scanty, concentrated urine.

Various **types of fever** are recognized, including:

1. *Continued fever.*—In which the daily variation in temperature is slight (1–2°).

2. *Remittent fever.*—In which the daily temperature variation is marked but the minimum is still above normal.

3. *Intermittent fever.*—In which the temperature variation is marked and falls to or below normal.

4. *Inflammatory fever.*—Symptomatic of some localized inflammation.

The **course of fever** is divided into:

I. The *onset*, or period of *invasion*, during which the temperature continues to rise.

2. The "*fastigium*," or period during which the temperature remains at its acme.

3. The period of *decline* or "*defervescence.*" In this stage death may be ushered in by either hyperpyrexia (above 105.5°) or collapse (below 96°); or the fever may terminate abruptly (*crisis*) or gradually (*lysis*) and convalescence be established.

The *typhoid, i. e., typhus-like state* is a condition present in many fevers. It is characterized by a dry brown tongue and sordes upon teeth and gums; feeble heart with weak, rapid pulse; faulty circulation, resulting in superficial inflammatory and destructive changes (bed sores), and muttering delirium, stupor, twitchings, tremor and sinking to the foot of the bed.

The **diagnosis of infectious diseases** depends upon the careful interrogation and physical examination of the patient.

A. The *interrogation* should embrace:

1. *A history of previous infectious diseases.* Yellow fever, scarlatina and measles usually confer immunity to a second attack, while rheumatism and pneumonia predispose to a recurrence.

2. *Possible cause of the present attack* (exposure to infection; general influence of patient's environment; and also any localized inflammation which may in itself have given rise to the pyrexia).

3. *The mode of onset* (prodromal symptoms, as in typhoid, or sudden invasion, as in scarlatina, cholera, etc.,) often affords a diagnostic clew.

B. *The physical examination* should include:

1. The *temperature.* This should be ascertained by insert-

ing a clinical thermometer into the mouth, axilla or rectum for a period of five minutes or more. The normal temperature in the mouth is 98.4° F.; that in the axilla is slightly lower, while in the rectum it is somewhat higher.

In investigating an infectious disease the temperature should be taken not once, but twice daily, at stated hours in the morning and evening. The variation determines the type of fever and assists both diagnosis and prognosis.

2. *Characteristic signs.* Inspection will reveal the various skin eruptions, etc., as well as aid in determining the condition of the sensorium (for example, the apathy of typhoid, meningitis and severe sepsis).

3. *The physical condition of heart, lungs, kidneys and digestive tract.* An exact knowledge of the state of these organs is of the utmost importance, for upon their integrity and their ability to bear the brunt of the attack recovery depends. Until this is ascertained a prognosis cannot be framed or a proper course of treatment directed.

The Management of an Infected Patient demands immediate attention from the physician. This implies:

1. *Isolation of the patient.* Let him be put to bed in a large upper room with good light and ventilation, all upholstery, curtains and unnecessary furniture being removed before the entrance of the patient. Allow no one else but the attendants to enter, and have each one take every precaution against conveying contagion from the room. The door should be screened with a sheet kept moist with some disinfecting fluid.

2. *Cleanliness.* The entire body of the patient should be cleansed with warm water and soap at least once daily. The mouth and teeth should be scrubbed frequently with a solution of boric acid or some other mild antiseptic. The bed clothing should be changed daily and must be disinfected before it leaves the room. All discharges should be received and allowed to remain for a time in vessels containing a germicidal solution. At the conclusion of the illness the patient and attendants should bathe, apply a disinfecting solution to

the body, don fresh clothing and leave the room immediately.

3. *Disinfection of the Room.* As soon as the room is vacated every inch of it should be cleaned with soap, water and disinfecting solutions. The paper should be removed from the walls and the woodwork repainted at once.

The Treatment of an Infected Patient consists, in great measure, of rest, careful nursing, a proper diet, and the treatment of individual symptoms as they arise.

Diet.—Patients must be individualized, but a majority of them thrive best on a strictly liquid diet. Good milk, administered in definite quantities at stated intervals (in adults, from two to four ounces every two hours) is best. Animal broths, eggs, and long-cooked gruels are occasionally desirable. Pure water should be given freely; and in cases presenting profound toxemia the latter may be supplemented by the introduction subcutaneously of large quantities of the normal saline solution.

Hydrotherapy.—At least one daily bath, with soap and warm water, is essential to cleanliness. In many of the infectious diseases the external use of water affords a simple and harmless means of reducing the temperature and controlling the extreme pyrexia. Among the more important methods for accomplishing this are the following:

The Sponge Bath.—The bed is protected by mackintosh or rubber sheeting, and a sponge or wash-cloth is saturated with water, the latter being at room-temperature or cooled with ice. Every portion of the patient's body is in turn bared, washed with moderate friction, and dried. In case the temperature is high it is well to dry the skin by fanning it rather than with cloths, thus aiding the abstraction of heat by evaporation. The sponging may be repeated every one, two, or three hours, according to the height of the temperature; it adds greatly to the comfort of the patient, but reduces the pyrexia so slightly, unless the water be very cold and applied frequently, that in severe cases it is wiser to resort to other methods.

Cold Compresses.—Three or four thicknesses of old table linen or toweling are moistened with cool water and applied to the body; and a second compress is applied as soon as the first one becomes warm. While agreeable to the patient, the compress has little effect upon temperature.

The "cheese-cloth bath," highly recommended by Goodno, consists of about four layers of cheese cloth dipped in ice water and then wrung out *almost dry*. The patient lies upon his side, a single layer of dry cheese cloth is placed over his body, and the moistened layers are laid over him so as to cover the body anteriorly, laterally, and posteriorly, from neck to knees. By this agreeable method heat is dissipated rapidly and the temperature quickly falls.

The Cold Pack.—A blanket is laid smoothly over the bed, and on it are spread one or more sheets wrung out in cold water. The patient is placed in the middle of the sheets and the latter quickly wrapped about him, and then the blanket is folded over all. At the end of half an hour the patient begins to feel warm and often perspires freely, though the temperature is considerably reduced. When it begins to rise again the pack may be repeated.

The cold bath, advocated by Brand and others and extensively used in Germany, must be instituted during the first week of the disease if at all. When the patient's temperature reaches 102.2° F. he is placed for about fifteen minutes in a bath tub containing water at 65–70°, and during this time he is rubbed by attendants. On entering the bath he receives about an ounce of whisky, well diluted, or half a glassful of wine. At the conclusion of the bath he is lifted to his bed, which is spread with a rubber sheet, and covered with a blanket. In ten or fifteen minutes he is thoroughly dried and his night dress replaced, and he is then given a cup of milk or broth. As long as the temperature remains high the bath is repeated every three hours. This method, while of undoubted efficacy, is little used in private practice.

STIMULANTS should be administered to an infected patient

only upon the presence of well-defined indications for their use, such as are afforded by progressive enfeeblement of the heart, high delirium, pulmonary consolidation, or great abdominal distention.

CONVALESCENCE demands great care in the resumption of ordinary habits of exercise, diet, etc. Solid food should be added to the dietary by degrees, and mental and physical exercise should be forbidden until the wasted body has had an opportunity to recuperate.

TYPHOID FEVER.

(Abdominal typhus; enteric fever.)

Etiology.—The *exciting cause* is the bacillus typhosus of Eberth. It is transmitted principally in drinking water contaiminated with the discharges from typhoid patients.

Autumn, early adult life and individual susceptibility constitute predisposing factors.

Pathology.—The essential lesions are:

1. *Changes in the intestinal lymphoid glands.* During the *first week* the solitary follicles and Peyer's glands become swollen and congested, with, perhaps, beginning ulceration.

During the *second week* ulceration continues and some sloughing takes place.

During the *third week* sloughing continues, leading sometimes to coalescence of adjacent lesions, or to perforation.

During the *fourth week* some ulceration and sloughing continue, but cicatrization begins, and ultimately there are left smooth depressed scars.

2. *Changes in the mesenteric glands* are similar to those in the intestine, but as the products of necrosis cannot be thrown off they form purulent foci which become encapsulated or absorbed on recovery. They may rupture into the peritoneal cavity.

3. The *spleen* becomes enlarged and hard.

4. The *toxemia* causes various complicating lesions, including the following:

(*a.*) The *liver* is enlarged and softened, and its cells may undergo granular and fatty degeneration.

(*b.*) The *kidneys* show parenchymatous degeneration, and occasionally acute nephritis or infarction occurs.

(*c.*) The *heart* muscle is degenerated, becoming soft and flabby.

(*d.*) The *respiratory tract* is the seat of catarrhal changes, while hypostatic congestion of the lungs is the rule and catarrhal pneumonia is common.

(*e.*) The *nervous system* and the glandular structures undergo inflammatory changes occasionally.

(*f.*) The general *muscular system* is degenerated.

Clinical Course.—After a *prodromal period* of a week or ten days during which the patient suffers with lassitude, headache, slight digestive disturbances, and often epistaxis, the *initial stage* is ushered in by a gradual, step-like rise of temperature day by day for a week. During this *first week* there is prostration, severe headache, and gastro-intestinal disorder.

The *second week* is marked by continued fever and aggravation of the existing symptoms. The tongue becomes dry and fissured; *rose spots* appear on the abdomen; the enlarged spleen is recognizable by palpation; and the headache gives way to mental apathy or mild delirium. The pulse becomes frequent; diffuse bronchitis is usually present; the abdomen is distended; there may be constipation or diarrhea; and prostration grows more and more profound.

The *third week* shows a temperature that is still high, but in favorable cases the morning remissions become more marked. The pulse is rapid and feeble, the heart's impulse weak; muttering delirium alternates with apathy; emaciation and prostration are extreme. There may be retention of urine or incontinence of feces. At this period there is danger of hemorrhage, perforation, and peritonitis, and pulmonary complications.

In the *fourth week* the temperature becomes normal and the symptoms subside.

Analysis of Symptoms.—Fever.—In typical cases the temperature rises gradually, being higher in the evening than in the morning, until at the end of a week it has reached 103° or 104°. During the second week it remains fairly stationary at the maximum, with slight morning remissions. In the third week the morning remissions become more marked, the evening maximum remaining unchanged. In the fourth week the

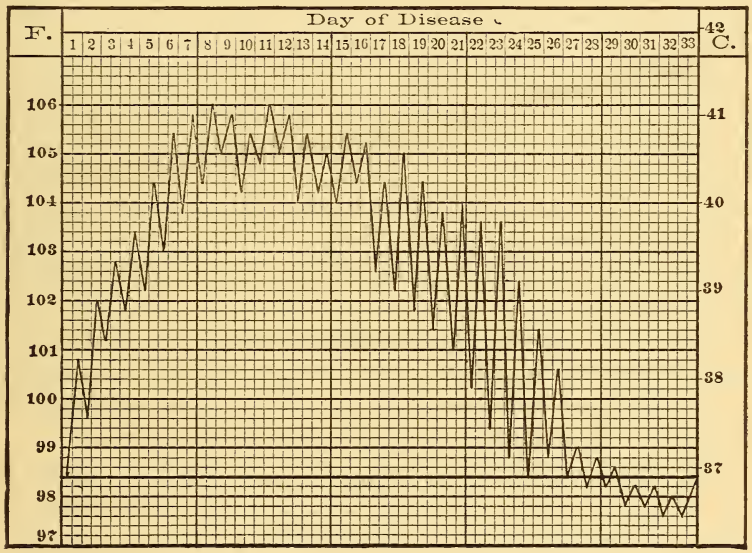

Fig. I. The Typical Temperature Curve of Typhoid Fever. (Salinger-Kalteyer.)

fever terminates by lysis, the morning temperature reaching normal or subnormal, and the evening rise growing less each day until it too is normal.

Variations from this typical course are common. In some cases the temperature rises quite abruptly at the onset, reaching its maximum on the second or third day. In some mild cases it may never rise above 101°, although if the other symptoms are severe this low temperature is not a favorable

sign. A rapid rise late in the course of the disease indicates the onset of a serious complication. A sudden fall suggests hemorrhage or perforation.

Circulatory Symptoms.—With the rise of the temperature the pulse becomes full and dicrotic, and at the end of the first week its frequency is about 100. During the second and third weeks it becomes quicker and feeble in direct proportion to the gravity of the case. The heart becomes weak, the first sound losing intensity until it resembles the second. The cardiac weakness permits a circulatory stasis which may result in hypostatic pneumonia, venous thrombosis, embolism, etc.

Nervous Symptoms.—During the first week headache is pronounced, and often is associated with sleeplessness and restlessness at night. In the second week these symptoms give way to mental apathy or delirium, the latter varying from a simple confusion of ideas to an acute mania. Later, delirium becomes profound, and with it there may be tremor, twitching (subsultus tendinum), and picking at the bed clothes or imaginary objects.

Respiratory Symptoms.—Bronchitis in some degree constantly accompanies typhoid; there may be rapid respiration, slight cough and bronchial rales. Lobular pneumonia is a less frequent complication, while lobar pneumonia is rare.

Abdominal Symptoms.—Pain and tenderness, especially in the right iliac fossa in association with gurgling on pressure, are symptoms generally present but by no means pathognomonic. Tympanites, due to paresis of the bowel, develops in the second week in most cases. Diarrhea, with stools like pea-soup, is often present, although at times constipation is persistent.

Hemorrhages from the bowels are of serious import; for the loss of blood, resulting from the erosion of vessels by the intestinal lesion, may be sufficient to destroy life at once. Hemorrhage is always a grave symptom, denoting extensive ulceration.

The eruption of typhoid is characteristic. It appears on

the abdomen or other parts of the trunk at the close of the first week, and consists of several small rose-colored macules, resembling flea bites, which disappear on pressure. Several crops appear on successive days. In some cases, particularly in children, the eruption is absent.

THE URINE presents the changes peculiar to the febrile state. At first it is reduced in quantity and of high color and specific gravity. Later in the disease, it is apt to contain a little albumen and a few casts, the latter indicating parenchymatous degeneration of the kidney.

Complications.—Any aggravated symptom, such as high temperature or excesive diarrhea, may be regarded as a complication. Among other complicating features, all of which occur most frequently in the third week, are intestinal hemorrhage, peritonitis, glandular abscesses, otitis media, venous thrombosis, nephritis, and pneumonia (catarrhal, rarely croupous).

Varieties.—*Walking Cases.*—These are mild cases in which the symptoms are so slight that the patient keeps about for a week or two. Nevertheless the intestinal lesions may progress rapidly, and grave symptoms, the result of perforation, may develop suddenly.

Abortive Cases.—This term is applied to cases which run a typical typhoid course for a week or two, and then all symptoms subside and convalescence ensues.

A Relapse is a second typical attack of typhoid immediately following a first and is supposed to be due to re-infection.

A Recrudescence is a sudden but temporary elevation of temperature, without aggravation of symptons, occurring during convalescence.

Diagnosis.—The majority of cases of continued fever met in this region, not due to pulmonary tuberculosis, are typhoid in character. It is well, therefore, to regard all doubtful cases as probably typhoid, reserving a positive diagnosis until the development of characteristic symptoms, such as the peculiar temperature curve, the enlarged spleen, and the eruption, establishes the identity of the disease.

Ehrlich's diazo-reaction, which appears in the urine about the close of the first week, may aid in diagnosis occasionally. Two solutions are required: (1) a 0.5% solution of sodium nitrite; (2) a mixture of 2 grams of sulfanilic acid, 150 c.c. hydrochloric acid, and 1000 c.c. of distilled water. One part of the first solution is mixed with forty parts of the second, an equal quantity of urine and about one-eighth volume of ammonium hydrate are added, and the whole is shaken well. To constitute a positive reaction both foam and fluid should become bright red in color. This reaction may be present in acute tuberculosis, meningitis, measles and other febrile affections; hence its diagnostic value is limited.

The *Widal serum test* yields a positive reaction in almost all cases of typhoid after the eighth day and often earlier. A drop of blood from a needle-prick of the ear or finger of the patient is allowed to dry upon a clean glass slide. A loopful of a fresh bouillon-culture of typhoid bacilli is placed upon a clean cover glass and to this is added a large loopful of a watery solution of the dried blood. The cover glass is inverted over the concavity of a hollow slide and sealed at the edges. Under a high power lens a rapid clumping of the bacilli can be observed in the hanging drop and their movement ceases almost instantly. If this reaction cannot be obtained at the end of a week typhoid can be excluded; but its appearance, on the other hand, may be the result of an attack ten years previously.

Acute miliary tuberculosis resembles typhoid very closely. The high remittent fever, the preponderance of pulmonary over abdominal symptoms, the consolidation at the apices rather than the base of the lungs, and the absence of eruption may serve to distinguish it.

Gastro-enteritis in children, with marked abdominal symptoms, may simulate typhoid. Greater fever, profound prostration, an enlarged spleen and the characteristic eruption indicate the latter, however.

Prognosis.—While generally favorable, the death-rate under

skillful treatment being less than ten per cent., the prognosis is influenced by three considerations, viz.,

1. *The severity of the infection.* Hyperpyrexia or profound toxemia and the resulting severe nervous symptoms are of unfavorable significance.

2. *Personal factors.* A history of family susceptibility, preexisting lesions of the heart, kidneys, or lungs, and surroundings which preclude proper sanitation and nursing, greatly lessen the probability of a favorable outcome.

3. *The presence or absence of complications.* Any severe complication renders the prospect much less hopeful. Heart weakness, in particular, is of serious import.

Treatment.—The treatment of typhoid fever resolves itself into four essentials, viz.,

1. *Absolute rest.* The patient must be put to bed under charge of a competent nurse and the slightest unnecessary muscular effort interdicted. The bed pan must be used, and even changes of posture must be accomplished with the aid of the attendant.

2. *Liquid diet.* Sterilized milk, two to four pints in twenty-four hours, and given in divided doses every two hours, is the best food. If this fails to be digested, as evidenced by the appearance of small coagula in the stools, peptonize it or add lime water, or change to koumiss, buttermilk or junket. Gelatin, beef tea and gruel may be useful at times. Sterilized water should be given freely.

3. *Sanitary precautions* to prevent the spread of the disease. This includes disinfection of the excreta. The latter should be allowed to remain in contact for an hour with a pint of a 1-1000 solution of corrosive sublimate, or of a solution of chloride of lime, six ounces to the gallon.

4. *Therapeutic measures* adapted to the individual. These may be considered as applied to typhoid in general and also as demanded by special symptoms or complications.

The pyrexia is best controlled by the use of external cold.

A certain point, about 102.5° F., may be regarded as a maximum, and elevation of the temperature beyond that point demands free sponging with cold water; and if the pyrexia persists, the use of the "cheese-cloth bath" or the cold pack. Hyperpyretic cases uncontrolled by these simple measures may be benefited by high colon flushes of cold water. The general cold bath, according to the method of Brand, must be used from the first days of the disease if at all.

Medicines.—During the initial stage of the fever the remedies most frequently demanded by the general condition of the patient are *bryonia* and *baptisia*. Less often special remedies are demanded by certain symptoms, such as headache (*belladonna*, *spigelia*, *gelsemium*) or general pains (*rhus tox.*, etc.).

With the advent of the second week the developing typhoid state generally demands *rhus tox*. At times a bad state of the mucous membrane will suggest *baptisia* or *mercurius*, and should either of these fail and the progress of the condition be evidenced by a red, raw tongue, with food badly borne, great thirst, and offensive watery stools, recourse may be had to *cantharis*, *lachesis* (especially if there be a comatose state) or *turpentine* (tympanites and other abdominal symptoms).

Catarrhal enteritis with mental apathy suggests *phosphoric acid*, while symptoms of gastric catarrh with constipation will be benefited by *hydrastis*.

Nervous symptoms, if profound, require another class of medicines, chief of which are *hyoscyamus* (furious delirium), *stramonium* (cheerful delirium), and *agaricus* (delirium with tremor and rigidity). Muscular failure with great prostration and low delirium calls for *hydrochloric acid*, while the extreme typhoid state, with heart failure and general collapse, may suggest *arsenic*.

Intestinal hemorrhage may be controlled by such remedies as *hamamelis* (sore feeling through the abdomen), *turpentine* (meteorism, flatulence, offensive stools, and possibly albuminuria), *hydrastis* (heavily coated tongue, etc.), and *ipecac* (griping colic below the navel). Should these fail and grave

danger be apparent, give *morphine sulphate*, gr. $\frac{1}{8}$–$\frac{1}{4}$, hypodermatically. Subcutaneous or intra-venous injection of a quart or more of warm saline solution is advisable if there be great loss of blood.

Tympanites may be controlled by care in diet, the application of cold to the abdomen, and a carefully selected general remedy. An extreme accumulation of gas may be relieved by the passage of a long rectal tube.

Perforation is treated as peritonitis, and in the absence of possible surgical intervention, is usually fatal. A rising leucocyte count in the presence of suspicious symptoms, may be made the signal for abdominal section.

Constipation should be relieved every second day by the use of an enema.

Stimulants are indicated when the first sound of the heart weakens and the pulse becomes soft. Good brandy or whisky, four to eight ounces in twenty-four hours, is best. *Strychnine sulph.*, gr. $\frac{1}{60}$–$\frac{1}{30}$, three to six times a day, may be required.

Heart failure, if advancing in spite of symptomatic treatment and stimulation, demands the hypodermatic use of *strychnine sulph.*, gr. $\frac{1}{60}$–$\frac{1}{30}$, *spartein sulph.*, gr. $\frac{1}{4}$, *agaricin*, gr. $\frac{1}{10}$, or camphorated oil (camphor, ʒj in olive oil, ℥j) in repeated doses of one drachm each.

Convalescence may be regarded as established after the temperature has remained normal for two or three days. It is better to wait at least a week, however, before giving any solid food, and then but little at a time. Any rise in temperature calls for an immediate return to liquid diet. Exercise must be attempted only as returning strength permits.

TYPHUS FEVER.

(Cerebral Typhus; Ship or Jail Fever.)

Etiology.—Typhus fever is endemic in parts of Ireland, England, and Continental Europe, occasionally becoming epidemic elsewhere. It is one of the most virulent of contagious

diseases, sparing few who come in contact with it. The infection is transmitted from person to person and by fomites, but its "striking distance" is limited to about five feet, the virus being destroyed by the air. The specific cause, presumably a micro-organism, has not been discovered. By preference it attacks those whose resistance has been destroyed by bad hygiene, starvation, filth, and overcrowding.

Pathology.—Typhus fever presents no distinctive lesion except its eruption. The general tissue changes are degenerative

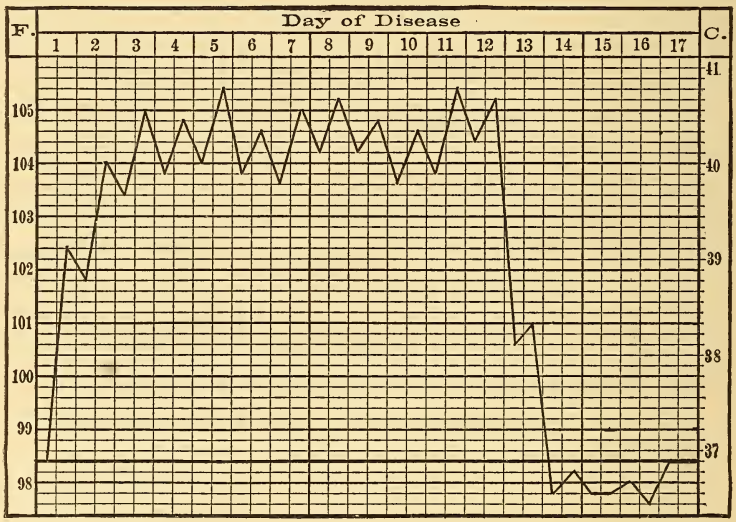

FIG. 2.—The Typical Temperature Curve of Typhus Fever. (Salinger-Kalteyer.)

in character and do not differ from those of typhoid fever and other acute infections in which toxemia is profound.

Clinical Course.—After few, if any, prodromes, there is an abrupt onset of chills or chilliness, followed by a fever, headache, nausea, vomiting, muscular pains and profound prostration. Within a day or two the temperature reaches 104° or 105° F. The face is flushed and the eyes injected; and about the fourth day the eruption appears. Delirium develops rapidly. At first it is mild, but soon becomes muttering or

maniacal, and in the course of the second week it may merge into coma or coma vigil (unconsciousness with open eyes). The typhoid state, with tremor, subsultus tendinum, dry, brown tongue, albuminuria, and incontinence of urine and feces, becomes fully developed; often it is complicated with bronchitis or broncho pneumonia. The temperature remains high continuously, and the pulse is rapid (120–140) from the beginning. The heart grows steadily weaker until in many cases a fatal issue is reached. Should this not occur, however, the febrile period ends by crisis about the end of the second week, when often the patient falls into a refreshing sleep from which he awakens convalescent.

The *eruption* appears from the third to the fifth day, and consists of dirty pink macules occurring on the covered portions of the body; these rapidly become petechial and do not disappear on pressure. Undeveloped spots give rise to a subcutaneous mottling, and the combined effect has been described as a "mulberry rash."

Complications.—Hypostatic congestion of the lungs; catarrhal pneumonia, possibly ending in gangrene; nephritis, and suppurative parotitis.

Diagnosis.—*Typhoid fever* is differentiated by (1) a gradual onset; (2) a less abundant, non-petechial eruption, which appears later in the disease; (3) the later development of nervous phenomena; and (4) the gradual termination.

Cerebro-spinal meningitis is marked by occipital headache, retraction of the head, hyperesthesia, and a tendency to opisthotonos and ocular palsies.

Prognosis.—The mortality varies from 10 to 50 per cent. in different epidemics. It is necessary in each case to consider (1) the severity of the infection, (2) the personal factor, and (3) the complications.

Treatment.—Isolation. Absolute rest. Liquid food. The power of atmospheric air to destroy the infection suggests the need of free ventilation, or even treatment in the open air. The general management, including hydrotherapeutic and

medicinal measures, does not differ from that required by typhoid.

RELAPSING FEVER.

Etiology.—The *exciting cause* is a spirocheta (*spirillum Obermeieri*), a slender motile spiral three to six times the diameter of a red blood corpuscle in length. It is found in the blood during a paroxysm, disappearing during the intermissions and

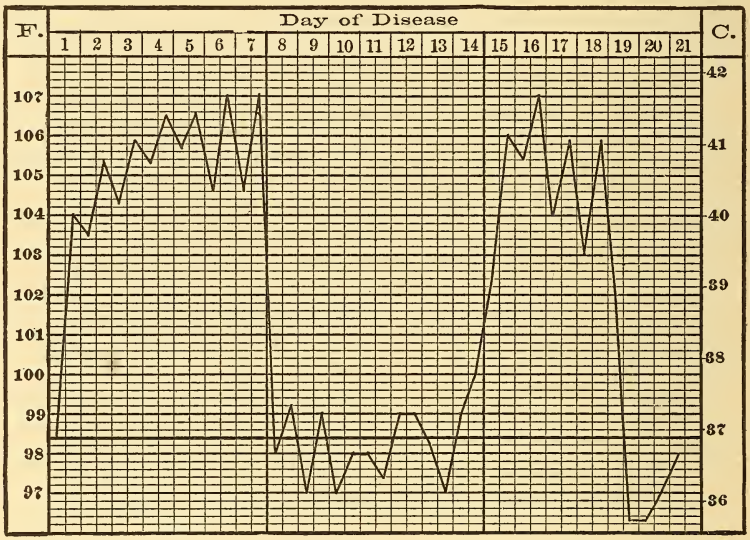

FIG. 3.—The Temperature Chart of a Case of Relapsing Fever. (Salinger-Kalteyer.)

giving place to round, glistening bodies supposed to be spores.

The disease is actively contagious, either through personal contact or by fomites. Overcrowding, filth, and starvation are predisposing factors, and epidemics often occur in connection with those of typhus.

Pathology.—There is no characteristic lesion. The spleen is enlarged and softened, and may rupture. Degenerative changes occur in the liver, kidneys, and heart.

Clinical Course.—The attack begins abruptly with a chill, followed by high fever (104°–105°) which remains continuous. The patient suffers with headache, severe general pains, and prostration, but the mind remains clear. Jaundice is common. About the fifth day the temperature falls suddenly to or below normal, and this crisis may be accompanied by severe sweating, exhausting diarrhea, collapsic symptoms, or hemorrhages from stomach, bowels, or kidneys.

After an intermission of five or six days there is a second but often milder paroxysm of the same duration. Generally convalescence then ensues, but the paroxysm may recur as often as five times.

Complications.—Hyperpyrexia; catarrhal pneumonia; nephritis.

Sequelæ.—Slow convalescence, often with muscular and joint pains and lymphatic swellings. Post-febrile ophthalmia, with possible loss of sight.

Diagnosis.—Any doubt will be removed by finding the spirocheta in the blood.

Prognosis.—Death, though not common (1–4%), may occur from cardiac thrombi or emboli, or from other complications.

Treatment.—The general management should be that of any infectious disease. The remedies indicated during the stage of invasion include *aconite*, *bryonia*, and *veratrum viride;* while the fully developed disease may require *bryonia*, *rhus tox.*, *baptisia*, *cimicifuga*, or *eupatorium perfoliatum.*

DENGUE.

(Dandy or Break-bone Fever.)

Etiology.—Dengue occurs epidemically in the tropics and sub-tropics during the summer season, frost dissipating the infection. It is contagious, but the specific organism has not been isolated.

Pathology.—The disease is essentially a toxemia accompanied

by an inflammation of serous structures (joints and tendon sheaths). No characteristic lesions have been found.

Symptoms.—The essential features of dengue are:

1. *Fever.* (*a*) An initial paroxysm lasting three or four days, followed by

(*b*) A remission of two or three days, and

(*c*) A terminal paroxysm, shorter and less severe than the first.

2. *Severe muscular and articular pains.* During the febrile paroxysm the muscles are tender and the joints are red, swollen and painful.

3. A *rash.* This is not distinctive: there may be an initial rash of scarlatinal character, which disappears quickly, and is followed on the fourth and fifth day by an eruption which may be erythematous, urticarial or herpetic. Often desquamation ensues.

Clinical Course.—The disease begins abruptly with a slight chill followed by fever (102°–107° F.), and headache, backache, and severe pains in muscles and joints. Prostration is marked and a scarlatina-like rash may be present. At the end of three to five days a crisis, often accompanied by diarrhea or sweating, occurs, and the general symptoms disappear, leaving the patient stiff and sore. At this time various eruptions appear. In two or three days the characteristic symptoms reappear, but this febrile paroxysm is milder and shorter than the first, and is followed by slow convalescence.

Less commonly the attack is accompanied by hemorrhages from the mucous membranes, inflammation of the respiratory tract, and lymphatic swellings.

Diagnosis.—*Rheumatic fever* runs a dissimilar course and is usually without eruption.

In *influenza* the temperature is not remittent, the joints are rarely involved, and serious complications are frequent.

Yellow fever may be distinguished by the occurrence of

jaundice, black vomit, albuminuria and severe nervous phenomena.

Prognosis.—Favorable; death is rare.

Treatment.—Rest in bed, generous fever diet, and such remedies as *aconite*, *bryonia*, *gelsemium*, *rhus tox*, *eupatorium* and *arsenic*.

MILIARY FEVER.

(Sweating sickness.)

Etiology.—An infectious disease, without definite pathologic lesion and with an unknown specific cause, which occurs in rare epidemics in districts of limited area. Usually it prevails in spring and summer and attacks a large percentage of the population in the invaded district.

Symptoms.—There are a few prodromal symptoms: headache, malaise and prostration. Then suddenly the patient is seized with moderate fever, profuse sweating and severe pain in the epigastrium. On the third or fourth day an eruption appears. It consists of small red spots, varying in shape and size, in the centre of each of which a vesicle forms in a few hours. The contents of the latter become opaque, and in the course of two or three days it dries up, forming a crust which falls off and leaves a normal surface. In severe cases profound nervous symptoms and asthenia may develop.

Diagnosis.—*Rheumatism* may be distinguished by its swollen joints; the eruption of *measles* is not vesicular; and *malaria* has a periodic pyrexia. Knowledge of the prevalence of an epidemic renders diagnosis easy.

Prognosis.—This varies with the character of the epidemic. The average death rate is about 10 per cent.

Treatment should be conducted as in other acute infections, therapeutic measures being adapted to symptomatic demands.

INFLUENZA.

(La Grippe, Catarrhal Fever.)

Etiology.— This disease occurs in great epidemics, which spread over the world with almost inconceivable rapidity. Sporadic cases occur at other times. It is mildly contagious, but on account of its rapid spread the method of infection is difficult to explain. Sex, age, condition and climate have no influence. The *exciting cause* is the bacillus of Pfeiffer, a minute rod found in the catarrhal discharges.

Pathology.—There are no characteristic lesions, the disease being essentially a profound toxemia.

Symptoms.—There is a sudden onset, usually with high fever which lasts for several days and is accompanied by profound prostration and great pain in the head, back, and extremities. As a rule the attack is marked by severe catarrhal conditions of the respiratory or gastro-intestinal tract.

Clinical Types.—The varying localization of the disease has led to its classification into:

1. *The Catarrhal Variety.*—This is distinguished by catarrhal inflammation of the *respiratory* or *gastro-intestinal* tract.

2. *The Nervous Variety.*—This form is characterized by delirium and hyperesthesia of the special senses. In addition a *typhoid type* may develop, in which the typhoid state is added to nervous and gastro-intestinal symptoms.

Complications.—These are extremely common and may involve the respiratory tract (catarrhal or croupous pneumonia, pleurisy, empyema), the circulatory system (endocarditis, pericarditis, heart failure), or the nervous system (neuralgia, neuritis, insanity).

Diagnosis.—The association of catarrhal symptoms with a *profound prostration altogether out of proportion to the severity of the other symptoms* renders diagnosis easy in most cases. Doubt may be removed by staining a cover-glass specimen of the respiratory secretion with Ziehl's carbol-

fuchsin or Lœffler's methylene blue, when the characteristic short, thick rods will be revealed. *Typhoid fever* may be distinguished by the Widal reaction.

Prognosis.—Influenza is rarely fatal except through complications, upon recognition of which the prognosis must be based.

Treatment.—Absolute rest in bed with a nourishing liquid diet. Stimulants are often necessary.

The symptoms at the onset are usually met by *gelsemium*, though sthenic cases may require *aconite*. Bronchial symptoms associated with intense pain in the extremities are relieved by *bryonia*. Great restlessness and pain suggest *rhus tox.*, while *eupatorium* is useful in cases in which inflammation of the upper respiratory tract is accompanied by marked pain and soreness of the entire body. Gastro-intestinal symptoms may demand such remedies as *ipecac*, *arsenic*, and *cuprum arsen.*

Complications should be treated as independent conditions.

YELLOW FEVER.

Etiology.—Yellow fever is endemic in the West Indies, whence occasionally it spreads along lines of travel to surrounding countries, particularly the southern United States. The disease is not directly contagious. The specific germ is probably conveyed from one infected individual to another by mosquitoes rather than by fomites. Thorough disinfection and quarantine are effective safeguards.

Pathology.—The invasion of the organism leads to:

1. *Rapid blood decomposition.* The red corpuscles are disorganized, hemoglobin is set free, and the blood becomes dark and its coagulability diminished.

2. *Tissue degenerations.* The toxemia leads to granular and fatty changes, and occasionally hemorrhagic infarcts, in the liver, kidneys, and heart. The gastro-intestinal mucosa becomes inflamed and eroded.

3. *Jaundice.* Often the skin becomes yellow, hence the name of the disease. In the early stages this may be slight, but later the patient's skin assumes a saffron-color.

Symptoms.—1. The *stage of invasion* is marked by a chill, a rapid rise of temperature, and pain in the head, back, and limbs. The face is flushed and the eyes suffused. The epigastrium is tender, the stomach is irritable, and vomiting occurs frequently. The temperature remains high for about three days, then falls to nearly normal.

2. The "*stage of calm*," the remission which now occurs, may initiate convalescence in mild cases. All the symptoms moderate, but as a rule, in a day or two, a third stage supervenes, viz.,

3. The *stage of relapse.* The temperature rises rapidly, jaundice develops, the symptoms become aggravated, and as a result of kidney involvement a uremic condition is added. There is intense gastric irritability and often "black vomit," due to the presence of altered blood in the vomitus. The urine is scanty, albuminous, and may contain hyalin casts and blood; complete suppression may occur. Cerebral symptoms develop, varying from mild delirium to profound coma. The prostration may merge into the typhoid state, or collapse may ensue. Hemorrhages beneath the skin and from the mucous membranes are common. The duration of this stage varies. Death may occur from collapse, hemorrhage, or uremia.

Diagnosis.—As a rule, the symptoms of yellow fever are so unmistakable that an error in diagnosis is impossible. A *malarial fever* can be excluded by the absence of the parasite from the blood.

Prognosis.—The mortality varies from 10 to 80 per cent., according to the epidemic. Hemorrhages, anuria, and cerebral symptoms are of grave significance.

Treatment.—Absolute rest. Little or no food during the period of invasion; later, light liquid food, and rectal feeding if necessary. Increasing prostration requires alcoholic stimulants.

Of remedies, in the early stage *aconite*, *belladonna*, and *camphor* have proved valuable. Later, gastric symptoms may require *bryonia*, or incessant vomiting may demand *ipecac* or *argentum nit.* The profound toxemia of the third stage calls for such remedies as *arsenic*, *lachesis*, and *crotalus*. Cerebral symptoms suggest *belladonna*, *hyoscyamus*, and *opium;* and the urinary symptoms are met by *cantharis*.

DYSENTERY.

Etiology.—Inflammations of the colon, for which dysentery is a generic term, are common in the tropics, with occasional sporadic or epidemic outbreaks in temperate regions. Their exciting causes, presumably microbic and supposed to be conveyed in drinking water, are not determined definitely. The amœba coli is credited with causing the tropical form.

The predisposing factors include a tropical climate, late summer and autumn, unhygienic surroundings, and dietetic transgressions.

Pathology.—The anatomical lesions permit a division into three acute forms, any of which may merge into a chronic form.

1. *Catarrhal dysentery.* The mucous membrane of the colon is swollen and congested and the solitary glands are enlarged and may ulcerate.

2. *Amœbic (tropical) dysentery.* The mucous membrane is edematous, with localized areas of cellular infiltration which become necrotic and slough off, leaving irregular ulcers. These undermine the mucosa, extending at times down to the serous coat; and they contain amœbæ in large numbers. Hepatic abscess is a common complication, and it may rupture into pleura or lung. A diphtheritic inflammation may be associated.

3. *Diphtheritic dysentery.* The colon is swollen and covered with a croupous exudate, which sloughs away and leaves large irregular ulcers.

4. *Chronic dysentery.* As a result of persistent inflammatory changes the walls become thickened and often ulcerated. Healing by cicatrization may lead to stricture of the intestine.

Symptoms.—After several days of diarrhea, which gradually becomes severe and painful, the stools lose their fecal character and instead consist largely of *blood*, pus and mucus. At the same time persistent and severe rectal *tenesmus* (bearing down) and *tormina* (abdominal griping) occur. There is moderate fever, considerable prostration, and in the diphtheritic form a typhoid state may develop. Death may occur from exhaustion or as the result of some complication, such as peritonitis or abscess of the liver. The duration of an acute attack varies from one to eight weeks, according to its severity.

Diagnosis.—Dysentery differs from ordinary *diarrhea* in the occurrence of mucoid and bloody stools associated with great tenesmus. *Local rectal conditions* may give rise to loose and bloody stools, but the history together with a local examination will reveal the true nature of the process.

Prognosis.—In ordinary catarrhal cases the prognosis is favorable, and in the tropical cases it is guardedly so, but in the diphtheritic cases the outcome is doubtful. In epidemics the mortality varies from 10 to 70 per cent.

Treatment.—Absolute rest in bed, with use of the bed pan, careful disinfection of the excreta, personal cleanliness, liquid diet, and stimulants when needed. Irrigations of the bowel with large quantities of boiled water, plain or containing ten grains of boric acid to the ounce, repeated every six to twelve hours, are advisable. Hot fomentations may be applied to the abdomen to relieve pain.

Of remedies, *mercurius dulcis* may be administered in mild attacks, but *mercurius corr.* is pre-eminently useful in tropical cases. In the severe forms *cantharis* and *arsenic* are frequently indicated also. Severe tenesmus may be relieved by *belladonna*, *aloes* or *nux vomica;* while colicky abdominal pain suggests the use of *ipecac*, *colocynth* or *cuprum ars.*

Care in diet and exercise during convalescence is imperative, lest the disease pass into a chronic form.

THE MALARIAL FEVERS.

Etiology.—The *specific cause* is a parasite (*plasmodium malariæ, hemosporidia*) which is conveyed to man by a mosquito (*anopheles*). The disease prevails in all parts of the world, but particularly in tropical and other warm climates. Low, damp and marshy regions are especially apt to be malarial.

Pathology.—The organisms appear in the blood as small, whitish protoplasmic masses of varying shape (discoid, annular, etc.), possessing rapid amœboid motion. Within a red corpuscle this small colorless body appears, and as it grows minute yellowish granules begin to develop. The corpuscle becomes somewhat expanded and loses its color, while the amœboid movements of the organism grow less and the amount of pigment increases and darkens. Finally the pigmentary granules collect into a clump, radial striations appear, and at last, the surrounding red corpuscle having become almost indistinguishable, the capsule ruptures suddenly and the fifteen or twenty segments, each a spore, escape into the blood. These spores invade fresh corpuscles, in which this cycle of development is repeated. The typical febrile paroxysm in the patient occurs at the moment of segmentation.

Several varieties of the organism, varying in form, cycle of development, and clinical manifestations, are recognized, viz.,

I. *The Parasite of Tertian Fever.* This organism, which has just been described, completes its life cycle in about forty-eight hours.

II. *The Parasite of Quartan Fever.* With the exception of a period of development extending over seventy-two hours, this organism differs from the tertian only in minor particulars, being smaller, less active, more refractive and showing a smaller number of segments.

III. *The Parasite of Estivo-autumnal Fever.* This is less apt to pass through the cycle of development in groups; organ-

isms in various stages coexist, and large ovoid and crescentic bodies are also seen. The precise period of time occupied by the life cycle of this parasite has not been determined. In connection with this and other varieties, extra-corpuscular forms, some with flagellæ, have been described.

By their action these organisms destroy the red corpuscles, liberating the pigment (*melanemia*) and ultimately cause extreme anemia. The spleen becomes greatly swollen (ague-cake), the liver moderately enlarged, and all the organs are discolored by the pigment.

Symptoms.—The *malarial paroxysm*, which occurs in the regularly intermittent forms, may be divided into three characteristic stages, viz.:

1. The chill.
2. The fever.
3. The sweat.

Prodromes are often slight and may be absent. If they occur, they consist of slight general lassitude and headache, and often yawning and stretching. There may be nausea and vomiting. Chilly sensations follow rapidly, and soon the patient is in the midst of a severe shaking chill. Usually this lasts for less than half an hour, during which period the temperature is rising steadily. Then the chilliness gradually diminishes and is succeeded by a sensation of intense heat. The skin, previously pale and cool, becomes flushed, hot and dry, the temperature may reach 106° or 107°; and aching in the head, back, and limbs becomes intense. After some hours of intense fever, sweating begins and is soon profuse, and then the temperature falls rapidly and all the symptoms are relieved. During the intermission which ensues the patient may feel quite well; but at the end of the period the attack is repeated just as before.

Types of Fever.—Malarial fevers are divided, according to the causative organism and the resulting periodicity of the paroxysms, into three main forms, viz.:

The regular intermittent forms:

1. *Tertian Fever.* This includes (*a*) tertian paroxysms; and (*b*) double tertian fevers, or a quotidian of tertian origin.

2. *Quartan Fever.* This includes the (*a*) quartan, (*b*) double quartan, and (*c*) triple quartan or quotidian of quartan origin.

The irregular form:

3. *Estivo-autumnal fever*, usually a tertian of malignant type, but possibly a quotidian or irregular continued fever. To this group belong all the pernicious and many of the tropical fevers.

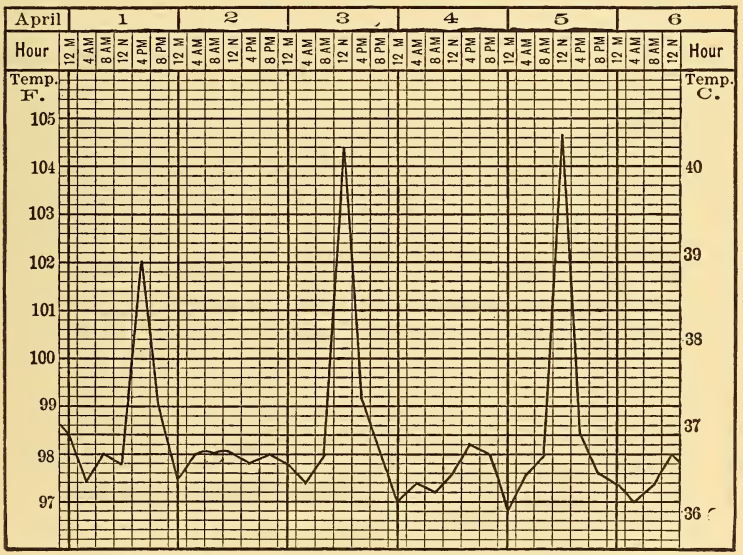

FIG. 4.—The Temperature chart of a Tertian Malarial Fever. (Salinger-Kalteyer.)

In addition to these, certain malarial phenomena have received special designations.

Pernicious or Malignant Malaria, rare in temperate climates but common in the tropics, is due to the abundance of parasites, their rapid multiplication and their malignancy, and to the special involvement of certain vital organs (central nervous system, gastro-intestinal tract, etc.). In consequence of the

latter fact, the pernicious paroxysm varies greatly in its clinical character. It may begin with violent delirium, which sometimes develops into mania or convulsions, occasionally into hemiplegia or symptoms of bulbar paralysis. More often the initial delirium merges into deep and often fatal coma. In some cases the symptoms simulate the algid stage of Asiatic cholera, while other cases are distinguished by hemorrhages (epistaxis, hemoptysis, hematemesis, and petechiæ) or by marked jaundice.

Malarial Hemoglobinuria occurs in connection with some severe malarial paroxysms, as the result of destruction of the red corpuscles by the parasites, together, perhaps, with the toxic action of quinine. The urine becomes an intense brownish-

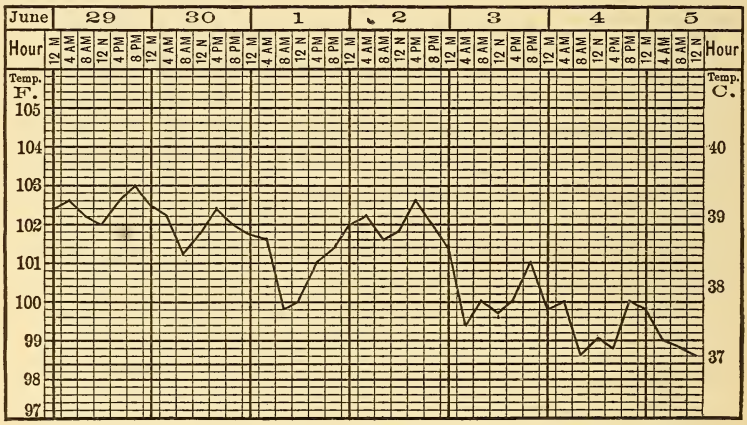

FIG. 5.—Temperature Chart of an Estivo-Autumna Malarial Fever. (Salinger-Kalteyer.)

black in color, and on standing deposits an abundant sediment of mucus, bladder and renal epithelium, pigment, and casts. Nephritis follows invariably.

Combined infections with different varieties of the parasite may occur. A double quartan infection, two groups of quartan organisms reaching maturity on successive days, occasions paroxysms on two days followed by one day of intermission. A triple quartan, three sets of parasites maturing on successive

days, causes quotidian paroxysms; or the latter may be evidence of a double tertian, two groups of the latter parasites maturing on alternate days.

Malarial Cachexia, resulting from repeated or long-continued attacks, is a condition of grave anemia. The skin becomes sallow, and there are severe headaches or neuralgias, marked dyspnea, gastro-intestinal disturbances and edema of the extremities. A *post-malarial anemia*, resulting from the extensive blood destruction, but less severe in type, is common also.

Complications.—The malarial attack may be accompanied or followed by pneumonia, pleurisy, tuberculosis, nephritis and typhoid fever.

Diagnosis.—The diagnosis of any form of malaria becomes positive only upon the demonstration of the plasmodium in the blood. For this purpose a drop of fresh blood may be examined under the microscope at the bed side or the drop may be spread upon a slide, allowed to dry in the air, and subsequently hardened in equal parts of alcohol and ether for half an hour and then stained with hematoxylin and eosin. Careful systematic search under a 1–12 inch lens will reveal the characteristic bodies within the red corpuscles.

In almost all conditions which simulate malaria a well-marked leucocytosis occurs. In consequence an increase in the number of leucocytes is strong evidence against the existence of an uncomplicated malarial fever.

The chronic malarial cachexia may be mistaken for primary or secondary *anemia*, *leucocythemia*, or *pseudo-leucocythemia*. In the absence of the parasite or of pigment in the blood, the diagnosis, except in leukemia, is difficult; but the history, together with the response of the patient to treatment, will ultimately distinguish the malarial case.

Typhoid fever is distinguished from malaria by its temperature curve, gastro-intestinal symptoms, and eruption. *Tuberculosis* is readily distinguished by its physical signs in association with the bacilli in the sputum. *Internal suppurations*

offer a history of previous disease, together with localized pain and physical signs. *Ulcerative endocarditis* may be excluded by auscultation over the heart. The intermittent fever of *cholelithiasis* is irregular, is associated with colic or pain in the hepatic region and jaundice, and ceases soon after the passage of the stone.

Prognosis.—Under appropriate treatment the outlook in the regularly intermittent fevers is good, and this statement applies, though less strongly, to the estivo-autumnal infection. Malarial hemoglobinuria is of grave portent. The malarial cachexia often requires for its control a change in the patient's environment.

Treatment.—If possible, remove the patient to a non-malarious district. Place him at rest in a room or bed protected by mosquito netting until the disease is controlled.

Quinine sulphate or *bisulphate*, in a dose of from ten to fifteen grains three or four hours before the paroxysms, acts as a direct parasiticide, destroying the organisms in the blood. It may then be continued in decreased dosage, the larger quantity being given only prior to an anticipated seizure.

Methylene blue, given in capsules containing one grain from three to six times daily, has a similar action and is especially recommended for the hemoglobinuria cases.

Pernicious paroxysms may be met by the intravenous injection of a solution of *quinine hydrochlorate*, gr. xv, and *sodium chloride*, gr. xij, in tepid distilled water, ʒiiss; or a similar solution may be used in larger doses hypodermatically.

In acute cases nausea may be controlled by *ipecac*, headache by *belladonna*, and a profound chill may suggest the use of *camphor*, *arsenic*, or *veratrum alb.*

The estivo-autumnal fever, if not controlled by *quinine*, may be benefited by the use of such remedies as *arsenic*, *quinine arsenite*, *arsenic iodide*, and *bryonia*. The cachexia should be combated by attention to general hygiene and nutrition, and the use of the remedies just mentioned, or possibly of iron preparations (*ferrum ars.*, *ferrum met.*, *ferrum phos.*).

RHEUMATIC FEVER.

(Inflammatory Rheumatism; Acute Articular Rheumatism.)

Etiology.—The *exciting cause* is a micrococcus, the infective agent entering the body through the tonsils, through wounds, etc. Among the more important predisposing causes are heredity, previous attacks, and exposure to wet and cold.

Pathology.—The articular changes consist of inflammation of the synovial membranes and ligaments, effusion into the joints, and inflammation and edema of the peri-articular structures, including tendon-sheaths, bursæ, muscles and fascia. The cartilages are roughened and may become eroded. The process usually terminates by resolution, but organization of plastic lymph may lead to fibro-cartilaginous ankylosis of the joint, and yet more rarely suppuration ensues.

Symptoms.—The invasion is usually abrupt, with a rise of temperature, sometimes preceded by a chill, and pain in certain joints.

1. The *joints* and surrounding structures are swollen, red, and painful. The medium-sized joints (knee, ankle, wrist) are first involved; the larger and smaller joints are attacked later. They are not affected simultaneously, the process changing from one joint to another from time to time; but anywhere from one to a dozen articulations may be inflamed at the same time.

2. The *skin* is covered with a copious, sour-smelling perspiration. Sudamina, erythema, urticaria, or purpura (*peliosis rheumatica*) may appear; and small subcutaneous nodosities (*rheumatic nodules*), which are firm, slightly movable, and painless, may develop in the tendons and fascia.

3. The *fever* varies in height according to the severity of the infection (101–105°), and hyperpyrexia (106–108°) occurs in some severe, generally fatal, cases. Restlessness, delirium, coma, and convulsions may accompany the febrile state.

4. The *heart* is quickened in its action, the pulse-rate reach-

ing 100 or more, and there is a notable tendency to the development of local infection in the form of endo-, peri- or myo-carditis. In children these lesions may occur in the presence of few if any articular manifestations.

5. *Blood changes* are marked, the red cells becoming greatly reduced in number, with a corresponding loss of hemoglobin, and the leucocytes becoming increased, even up to 39,000. Fibrin and lactic acid are in excess.

Course.—Indefinite. The duration of the disease may vary from a few days to many weeks, and it may merge into a subacute form.

Complications.—Endocarditis (very common), pericarditis, myocarditis, hyperpyrexia, pleurisy, pneumonia, chorea, iritis, meningitis.

Diagnosis.—*Gout* occurs in older persons, affects small joints, the fever is not high, and there are no acid sweats.

Osteomyelitis gives a history of long-continued pain, worse at night; it affects generally but one joint and presents moderate fever and no acid sweats.

Septic arthritis occurs in connection with some septic process elsewhere, its onset is slower, it has no acid sweats, and tends to end in suppuration.

Prognosis.—While recovery is the rule, complications may render the outlook grave; and the persistence of the valvular heart disease induced by endocarditis may seriously affect the future health of the patient.

Treatment.—Place the patient at absolute rest in bed between blankets, and wrap the joints in absorbent cotton. Cleanse with warm water and soap daily, nourish with semi-liquid food, and allow plain, ærated, or acidulated water freely. Examine the heart daily for signs of its involvement. *Aconite* may be given early in restless, apprehensive patients with active fever. As the disease progresses, *bryonia* is demanded by pains which the slightest motion aggravates, and which may be accompanied by thirst, gastric irritability, constipation, etc.; pericarditis, pleurisy, or pneumonia is apt to afford

additional indications for its use. *Rhus tox.* is required in cases so restless that the patient must move in spite of the pain, and the latter may be general, involving back and limbs as well as joints. *Pulsatilla* is recommended for cases in which the disease wanders from joint to joint, or especially affects the knees; *mercurius*, when the patient sweats freely, has aggravated pain at night, and presents symptoms of gastro-intestinal catarrh. *Belladonna* is suggested by red, throbbing joints and characteristic general symptoms; *caulophyllum*, by involvement of small joints, and *cimicifuga* and *phytolacca* by involvement of the trunk muscles. In typical cases a solution of *colchicine*, one grain to the ounce of alcohol, may be given in doses of 3–5 drops every two to four hours, and the effect is often magical. *Sodium salicylate*, in doses of 5–15 grains every two hours until pain is relieved and then in lessened dosage, is widely used. In every case the patient must remain at rest, carefully protected from the slightest exposure, for some days after every symptom has disappeared.

CEREBRO-SPINAL FEVER.

(Epidemic Cerebro-spinal Meningitis, Spotted Fever.)

Etiology.—The disease may be epidemic or sporadic. The *exciting cause* is probably the meningococcus intracellularis. Cold weather and bad hygiene favor its development.

Pathology.—The disease process begins as a congestion of the cerebro-spinal membranes, followed by the appearance of an exudate that is first serous and then purulent. The cortex and the nerve sheaths become involved in the inflammatory process; while the toxemia causes degenerative changes in the viscera.

Symptoms.—The onset is sudden, and is accompanied by chill, fever, prostration, *headache* and general pain. *Vomiting* may occur early or late and is often obstinate. More suggestive of the nature of the disease, however, is the severe headache with *stiffness of the neck* (from irritation of the upper cervical nerve roots). The symptoms grow worse rapidly, and *de-*

lirium, and in severe cases coma, supervenes. In connection with the mental changes there may be *signs due to the involvement of certain regions*. Thus, pressure upon the cranial nerves may produce strabismus, nystagmus, ptosis, pupillary disturbances, optic neuritis with destructive changes in the eye, trismus and deafness. Pressure on the spinal nerves causes intense cutaneous *hyperesthesia*, stiffness and twitching of muscles, and occasionally swollen and painful joints. Opisthotonos, with constipation and retention of urine, may ensue. Death may occur within a few days.

The temperature is irregular; leucocytosis is marked; and various atypical eruptions appear (herpes, erythema, urticaria, petechiæ). The course of the disease varies from one to many weeks.

Complications and Sequelæ.—Pneumonia (croupous or catarrhal), pericarditis, endocarditis and pleurisy occur as complications. The destructive changes in the nervous tissues may lead to incurable headaches, mental enfeeblement, paralyses, blindness, and deafness.

Diagnosis.—The coincidence of headache, vomiting, nuchal rigidity and hyperesthesia of the entire nervous system strongly suggests cerebro-spinal meningitis. It may be necessary to exclude *typhoid fever* (gradual onset, with absence of herpes and of leucocytosis), *pneumonia* (physical signs and sputum), *abscess of the brain* (localized symptoms, no rigidity or hyperesthesia), and *tubercular meningitis* (tuberculosis elsewhere, gradual onset, basal symptoms).

Kernig's sign, said to be pathognomonic of cerebro-spinal meningitis, consists of a flexure contraction of the knee-joint, which in the sitting posture cannot without violence be straightened beyond 135° with the thigh, but which is easily straightened when the patient is erect or recumbent.

A question as to the causative organism may rarely render lumbar puncture advisable. A long needle is passed a little to one side of the median line between the third and fourth lumbar vertebræ in adults, or between the fourth and fifth in

children. The escaping fluid may be examined microscopically and in cultures for the meningococcus intracellularis, the tubercle bacillus, etc.

Prognosis.—The outlook is bad but not hopeless. The mortality varies from 20 to 75%. Severe cerebral symptoms render the prognosis grave.

Treatment.—Rest and quiet in a somewhat darkened room, and a nutritious diet, liquid during the height of the disease, but semi-solid (rice, eggs, milk toast) as soon as convalescence begins. Stimulants are frequently required.

For the early inflammatory symptoms, *belladonna*, *bryonia*, *gelsemium*, and *veratrum viride* are recommended. For the fully developed case *cuprum acet.* (marked cerebral symptoms), or *cicuta* (predominating spinal symptoms) is to be preferred. Cerebral cases with coma may derive benefit from the use of *apis*, *opium*, or *helleborus*. A typhoid state will suggest the administration of *arsenic*, *rhus tox.*, *baptisia*, *crotalus*, or *lachesis*. *Actea* is recommended for pains and spasms continuing after the acute symptoms have subsided.

PNEUMONIC FEVER.

(Croupous Pneumonia, Lobar Pneumonia.)

Etiology.—The *exciting cause* is a bacterium, generally the pneumococcus (micrococcus lanceolatus), but possibly at times other organisms, including the bacillus pyogenes, the influenza bacillus, and Friedlander's bacillus.

Predisposing factors, which tend to lessen resistance to infection, are exposure to cold, the extremes of life, traumatism, and prior attacks of the disease.

Symptoms.—The onset is sudden, and is generally attended by *chill*, *high fever*, *pain in the side*, and *cough*. The respirations become very rapid (40–80 per minute) and *out of proportion to the pulse rate*. Dyspnea may be marked. The *face is flushed*. The *sputum* is "rusty" or blood stained; less often, it is dark like prune juice. The gastro-intestinal system is deranged, the

spleen is enlarged, chlorides disappear from the urine, and there is a marked leucocytosis. Herpes appear upon the lips. Headache is common, and patients suffering from typhoid, nephritis or alcoholism may become delirious. The fever is continued for from three to fourteen days, and then terminates by crisis in most cases, resolution and convalescence ensuing. Resolution may, however, be delayed for weeks, the temperature declining gradually.

In children pneumonia is frequently ushered in by convulsions, followed by delirium and stupor. In the aged it may develop insidiously, with little fever and few symptoms except profound prostration. Cough may be absent in young or old.

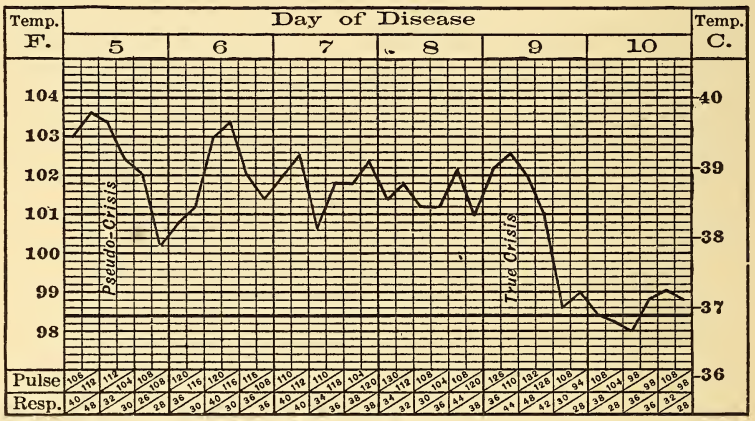

FIG. 6.—Chart Showing the Temperature Range in a Case of Pneumonic Fever. (Salinger-Kalteyer.)

Pathology.—The process consists of a fibrinous exudation, with subsequent coagulation, into the alveoli of one or more lobes of the lung. Three stages are distinguished, viz.,

1. The *stage of congestion.* The portion of lung involved is intensely congested, dark red in color, heavier than normal, and from its cut surface frothy, blood-stained serum exudes; but since the air cells are not yet occluded, it crepitates on pressure and a fragment will float in water. Microscopically,

the capillaries of the alveolar walls appear distended with blood, and the air cells contain exfoliated epithelial cells and blood corpuscles.

2. The *stage of consolidation.* The tissue becomes solid and a mottled reddish brown in color, resembling liver tissue (*red hepatization*). It tears easily, and since it has become airless it no longer crepitates and will sink in water. Microscopically, the air cells appear distended and packed with clotted fibrin in whose meshes are held red blood corpuscles, leucocytes, epithelial cells and micro-organisms.

3. The *stage of resolution* or diffuse suppuration. The red color gives place to gray (*gray hepatization*), this being due to the pressure of the exudate, the destruction of the red corpuscles and the appearance of many leucocytes. Resolution begins in spots, in which fatty degeneration and liquefaction of the exudate occurs and is followed by its absorption or expectoration. In unfavorable cases, however, the lung may become infiltrated with pus, with consequent abscess or gangrene.

Pleurisy, localized over the affected lung, is the rule (pleuropneumonia). The toxemia accompanying the local lesion, sometimes profound, leads to degenerative changes in the heart and other viscera. In addition, complicating lesions may occur as the result of infection of other organs (pericarditis, endocarditis, meningitis).

Physical Signs.—*First stage* (congestion): tympanitic, slightly dull note over the affected (usually lower) lobe; crepitant rales.

Second stage (consolidation): complete dulness; bronchial breathing; increased vocal fremitus.

Third stage (resolution): the dulness gradually clears up, the bronchial breathing becoming vesicular, with numerous moist rales.

Diagnosis.—The sudden onset, the characteristic sputum and the presence of leucocytosis, in connection with the physical signs, render diagnosis easy in most cases. The frequent

occurrence of pneumonia late in the course of chronic diseases (diabetes, nephritis, etc.), without marked symptoms, suggests the need of careful physical exploration of the chest in all doubtful cases.

Pleurisy lacks the abrupt onset, the peculiar sputum, and the change in the pulse-respiration ratio. In pleurisy vocal fremitus and resonance are diminished, the breath sounds are distant, and the upper line of dulness changes with the patient's posture.

Pulmonary edema presents neither chill, fever, nor pain; the sputum is watery, and auscultation reveals moist rales over the entire area of both lungs.

Pneumonic phthisis may be distinguished by irregular fever continued beyond the usual period of a pneumonia, and by a consolidation which begins more often at the apex than the base, and which subsequently presents signs of softening and excavation. It is accompanied by increasingly grave symptoms, and by tubercle bacilli in the sputum.

Typhoid fever is distinguished by a gradual onset, with abdominal symptoms and a characteristic rash; pneumonia appears as a complication late in the disease.

Prognosis.—The average death rate is about 20 per cent., but with careful treatment amidst favorable surroundings this can be greatly diminished. High fever, profound toxemia, the involvement of more than one lobe, and heart weakness are unfavorable conditions.

Treatment.—The patient should be placed at absolute rest and his diet should consist solely of liquids, especially if the temperature be high. The surface of the body should be sponged frequently. Excessive local pain may be relieved by a hot poultice. Hyperpyrexia may require cold sponging or the use of the ice pack. Heart weakness demands the use of stimulants; alcohol, strychnine, or camphor.

In the hyperemic stage, sthenic cases may be modified by the administration of *aconite* or *veratrum viride;* asthenic patients, especially those already feeble and debilitated, derive

greater benefit from *ferrum phos.* At any stage of the disease marked pleurisy will suggest the use of *bryonia.*

With the development of consolidation, the symptoms may suggest the use of *phosphorus.* This remedy is of especial value in patients of middle or advanced age, in whom the pneumonia occurs secondarily or in association with typhoid symptoms. *Sulphur* is indicated in pneumonias uncomplicated by pleurisy or bronchitis, and occurring at the extremes of life.

In the stage of resolution, cases associated with marked bronchitis and diffuse moist rales are benefited by *antimon. iodid.* Purulent infiltration suggests *stannum iodid. Tartar emetic* is demanded by marked pulmonary failure, with edema, loud rales, and cyanosis. *Antimon. arsen.* may be used under similar conditions.

The development of a typhoid state will require the administration of such remedies as *agaricus*, *arsenic*, *baptisia*, *bryonia*, *hyoscyamus*, *phosphoric acid*, and *rhus tox.*

ASIATIC CHOLERA.

Etiology.—The *exciting cause* is the comma bacillus, which gains entrance with food or drink, and is thrown off with the intestinal discharges. The disease is endemic in India and thence spreads along lines of travel to become epidemic in other counties.

Pathology.—There is no specific lesion. A catarrhal inflammation of the mucous membrane of the intestinal tract is associated with a serous drain which causes the blood to become thick and dark, while all the tissues become dry and the body appears shrivelled. The disease is essentially a poisoning of the system with the toxins of the comma bacilli.

Symptoms.—The attack begins with *diarrhea* accompanied by prostation and gastric disturbances. In some cases the disease is controlled at this early stage and recovery ensues in a few days (*cholerine*). Otherwise, the diarrhea becomes a

copious purging. The stools soon lose their fecal character and are composed of frothy serum containing flakes of desquamated intestinal epithelium—the "*rice water*" *stools.* The passages are usually painless, though griping may occur. At this stage severe and persistent *vomiting* appears; first the stomach contents are thrown off, then "rice water;" and severe *muscular cramps*, with retention of urine and the phenomena of collapse, ensue. The surface of the body becomes cold, shrivelled, and covered with clammy perspiration. The surface temperature is depressed, although internally it may be elevated. The pulse becomes rapid, thready, often imperceptible. This constitutes the "*algid stage*" which lasts for several hours and often ends in death. Consciousness may be retained almost to the end. In favorable cases the vomiting and diarrhea gradually cease, collapsic symptoms disappear, warmth returns, urine is again secreted, and this *stage of reaction* is followed by convalescence.

Cholera runs a rapid course, most of the fatal cases terminating within 24 hours. In patients who recover, convalescence may be prolonged over two or three weeks by **complications,** such as nephritis, pneumonia, pleurisy or multiple abscesses.

Diagnosis.—Doubtful cases of cholera may be diagnosed by the recognition of the comma bacillus in stained preparations of mucous floccules from the dejecta, by the behavior of cultures, or by the agglutination test,

Prognosis.—Guarded. The mortality averages 50 per cent.

Treatment.—Prophylaxis may be attained by quarantine, personal cleanliness, and care in boiling the drinking water and cooking the food.

Infected patients should be put at rest and use of the bed pan enforced. All discharges must be disinfected. Withhold food until the symptoms are controlled. Cracked ice or iced champagne may be given to assuage thirst; and abdominal pain may be relieved by hot applications. Hot general baths aid in controlling the severe muscular cramps and stimulate the circulation. The extreme loss of fluid may be compen-

sated by the intravenous or hypodermatic use of saline solutions.

Of remedies, administer *camphor* in drop doses on sugar during the initial stage. When diarrhea, vomiting, cramps, and failing circulation appear, *cuprum*, *cuprum arsen.*, or *verat. alb.*, should be given. Later, when reaction is established, *baptisia*, *bryonia*, or *rhus* may be indicated; or the suppression of urine and symptoms of nephritis will afford indications for the use of *cantharis*, *rhus tox.*, or *terebinth.*

DIPHTHERIA.

Etiology.—The *exciting cause* is the Klebs-Lœffler bacillus. Secondary infection with pyogenic cocci may occur. The disease is endemic in the large towns, occasionally becoming epidemic. Childhood amidst unhygienic surroundings favors infection.

Pathology.—The bacilli find lodgment upon the mucous membrane of the tonsils, pharynx, larynx, trachea, or nares (rarely that of intestines or vagina), and give rise to a fibrinous exudate with necrosis of the epithelial structures (diphtheritic membrane) which extends to contiguous parts. Mechanical interference with respiration and deglutition ensues; and at the same time a violent toxin is thrown into the circulation and occasions rapid degenerative changes in the viscera. The accompanying pyogenic cocci may be absorbed, thus adding a pyemic factor to the disease.

Symptoms.—These are *local*, varying according to the location of the lesion, and *general*, due to the violent toxemia.

Local.—More or less local soreness or pain is complained of, especially if the lesion be in the throat; and examination discloses at first a hyperemia of the mucous membrane, soon followed by the appearance of an *exudate*. The latter may develop in one spot or in a number of patches which rapidly coalesce; it is grayish or yellowish in color, and attempts to remove it expose a raw, bleeding surface beneath. Under the

microscope it is seen to consist of leucocytes, bacteria, and degenerated epithelial cells in a mesh of fibrin.

As a rule, the disease, if it invades the nasal cavities or the larynx, spreads to them from the throat; but it may occur primarily in either. Nasal diphtheria is usually attended by a purulent or bloody discharge and grave constitutional symptoms, and examination reveals the false membrane on the nares. Laryngeal diphtheria (*membranous croup*) is accompanied by hoarseness, croupy cough, and gradually increasing dyspnea and cyanosis. False membrane may be expectorated.

General.—The invasion is usually insidious, the symptoms being those of other *febrile* attacks, *i. e.*, headache, lassitude, and digestive disturbances. The temperature is not high, and is often irregular. If the disease is not controlled at the outset, profound systemic poisoning leads to severe *nervous symptoms* (delirium, stupor, coma), or to sudden or gradual heart failure. Erythematous or purpuric skin eruptions may appear. Albuminuria is frequent, but actual nephritis is rare.

Complications.—Extension of the membrane to the bronchi and lungs occurs in many fatal cases. Hemorrhages may result from the ulceration and erosion of mucous surfaces. Paralysis, due to a toxic peripheral neuritis, is a common complication or sequel: it may involve the nerve supply to the heart, the soft palate, the eye muscles, the larynx, or other portions of the body.

Diagnosis.—The discovery of the membrane usually suffices for diagnosis. Occasionally swelling of the glands at the angle of the jaw, accompanied by grave constitutional symptoms, precedes the appearance of the false membrane in the throat. In doubtful cases it is advisable to submit a culture to expert examination for the bacillus.

Follicular tonsillitis may closely resemble diphtheria and any doubt requires its treatment as a case of the more serious disease. As a rule, however, the exudate of tonsillitis can be seen to have orginated in the crypts, it is limited to the tonsil, and its removal is easy and reveals uninjured mucous membrane beneath.

Scarlatina sometimes presents a pseudo-diphtheria, a phlegmonous throat-lesion due to streptococcic infection. The sudden onset and typical rash serve to distinguish the disease.

Prognosis.—Guarded. The former mortality averaged 25%, but by the use of antitoxin this has been greatly reduced. Extension to nares or larynx and evidences of profound toxemia render the outlook more grave.

Treatment.—Isolation, rest, and nutritious liquid diet. Administer at once 1000 to 2000 units of *antitoxin*, according to the age of the patient. Should improvement, evidenced by lessening pyrexia and checking of the spread of the membrane, fail to appear in six hours, repeat the injection; and continue to do so at similar intervals until the disease is controlled. The injections may be made into the buttocks or the subscapular spaces.

Previous to the discovery of the specific antitoxin, much good was accomplished by the use of such medicines as *apis*, *lachesis*, *mercur. corros.* and *cyan.*, *cantharis*, *rhus tox.*, and the potash salts (*kali bich.*, *carb.* and *chlor.*).

Sprays of boric acid or hydrogen dioxide (1–10) may be used to cleanse the throat. Laryngeal stenosis may demand intubation or tracheotomy for its relief. Profound prostration or heart failure requires the administration of alcoholic stimulants.

Post-diphtheritic paralysis is benefited by such remedies as *gelsemium*, *cocculus* or *chloride of gold*, together with the use of the galvanic current. The anemia of convalescence affords an indication for the use of *arsenic iodide* or the various iron combinations.

ERYSIPELAS.

(St. Anthony's Fire.)

Etiology.—The *specific cause* is the streptococcus pyogenes, which gains entrance through some break in the integument. Among predisposing causes are the various cachexias, debility, wounds, and contact with infected cases.

Pathology.—This disease is a specific inflammation of the skin. It varies from an involvement of the superficial strata to an intense phlegmonous change extending to the subcutaneous structures, which are filled with serum, leucocytes, and streptococci.

Symptoms.—*Local.*—The usual point of invasion for the streptococci is about the head, especially the nose, face, or ear; but they may find entrance at any part of the body At this point on the skin *redness* and *swelling* appear, together with smarting and burning sensations. The inflammation spreads visibly from hour to hour, a well-defined *elevated margin* separating it from the normal skin. Vesiculation and suppuration, and even gangrene, may occur in connection with the inflammatory process. The disease may involve the mucous tracts with serious results. As a rule, the area involved is limited, the disease being controlled within a week or two; but in the so-called "ambulatory" cases the inflammation may continue to spread until each portion of the body surface has been involved in turn.

General.—Upon invasion, even before local changes have become manifest, the patient is seized with a *chill*, and this is followed by irregular, often high *fever*, *pain* in the head and extremities, and digestive disturbances. At the cessation of the local inflammation the temperature drops to the normal. In severe cases the toxemia may give rise to profound prostration and dangerous cerebral symptoms, while by its dissemination the streptococcus may occasion meningitis, endocarditis, peritonitis, or pneumonia, and in phlegmonous cases septicemia or pyemia.

Diagnosis.—*Erysipelas* may be differentiated from a simple *erythema* by its pyrexia, marked systemic disturbance, and the elevated margin of the spreading inflammatory lesion. Acute *eczema* presents marked itching, redness, and swelling without margin, and fever is absent.

Prognosis.—While recovery is the rule in uncomplicated erysipelas, in the case of a debilitated patient the prognosis must be guarded.

Treatment.—Rest. Nutritious food given frequently in small quantities; if much fever, liquid diet. Profound prostration may demand alcoholics.

To the local lesion apply cold water or cloths wrung out in ice water. Antiseptics, such as an ointment of icthyol and lanolin (20%), or a weak solution of carbolic acid (2%), may be applied with benefit, especially in recurrent cases. Suppurations and abscesses must be treated without delay in accordance with general surgical principles.

Of internal remedies, *belladonna* is suggested by smooth red swelling without vesiculation. Sthenic cases may require *aconite* or *veratrum viride*. If vesicles appear, *rhus tox.*, or less often *anacardium* or *croton tig.*, will be indicated, while marked edema will suggest the use of *apis mel.* Profound toxemia with delirium or coma demands such remedies as *hyoscyamus*, *stramonium* and *lachesis*. Suppuration is met by the use of *hepar*, *mercury* or *arsenic*, and recurrences suggest the need of *sulphur*.

SEPTIC FEVERS.

Etiology.—Septic organisms and their ptomains may enter the system:

1. Through wounds, surgical or traumatic;

2. Through the uterus, or tubes, following labor or abortion;

3. Through the mucous membranes, as a result of tonsillitis, typhoid, etc., or from the presence in the bowel of foods containing ptomains (*sepsis intestinalis*);

4. As a result of suppurative bone disease.

SAPREMIA. (*septic intoxication*) is an intoxication produced by the saprophytes of putrefaction, the material being contained within a more or less closed cavity with absorbent walls. Notable examples of this are a putrefying placenta or a blood clot within a wound or within the abdominal cavity. The infection is often a mixed one, and at times it is difficult to distinguish the condition from septicemia. The effects vary

in proportion to the amount of necrosed tissue, and the rapidity of absorption: it may kill in a few hours; it may be checked by the exhaustion of the pabulum upon which the saprophytes depend for their sustenance and growth; it may be cured quickly by their removal; or the putrefaction may be followed by suppuration.

Symptoms.—At the outset there may be slight pyrexia, with malaise, headache, coated tongue, and anorexia. This is followed by a chill, high fever, a rapid, soft pulse, vomiting, diarrhea, scanty urine, restlessness, delirium, jactitation, and cold sweats. Finally the pulse becomes weak, defecation and micturition are involuntary, the patient becomes comatose, and death ensues.

Treatment.—Asepsis and antisepsis afford prophylaxis. Should the symptoms of sapremia develop, the surgical indications are to relieve pressure, and to evacuate and drain the cavity, frequently using irrigation. In severe cases the use of purges and enemata, together with hypodermatics of strychnine, may be necessary. Among the remedies frequently indicated are *arsenic*, *carbo veg.*, and the *snake poisons*.

SEPTICEMIA (*septic infection*) is a toxemia due to the spread of pyogenic bacteria throughout the system and a progressive intoxication with the products of these bacteria. The living bacteria are present in the blood, where they reproduce themselves; and as a result, *e. g.*, in post mortem or dissecting wounds, rapid systemic infection may take place before the development of marked local lesions (primary septicemia). As a rule the development of septic germs requires a longer period than does the process of putrefaction, and therefore sapremia occurs earlier than septicemia.

Symptoms.—A variable period of incubation is followed sometimes by a chill, always by a rise of temperature, headache, anorexia, alimentary disturbances, and a typhoid state. The infection spreads by way of the lymphatics, whose course is marked by the red line of lymphangitis; the glands are inflamed (lymphadenitis), and the spleen and bone marrow be-

come involved. *Diarrhea* and faint *jaundice* are common; and various skin eruptions, erythematous or the result of thrombosis of the dermal capillaries, may appear. Leucocytosis is present. Degeneration of the heart muscle is indicated by the rapid and weak pulse; and endocarditis or pericarditis may supervene. The skin becomes cold and clammy, the patient is prostrated and indifferent, colliquative diarrhea sets in, the urine becomes scanty or suppressed, and finally there come delirium, stupor, coma, and death. Local symptoms are present at the point of infection: there is edema and redness of the edge of the wound, with an offensive discharge or actual sloughing. The microscope reveals capillaries filled with infected thrombi, and the vessel walls and lymph spaces are infiltrated with micro-organisms.

Treatment.—*Local.* Find the cause: open the wound, evacuate the clot, cut or scrape away the sloughs, cauterize living tissue, even amputate if necessary. Hot water or antiseptic poultices may be used locally.

General. Support the patient's strength with nourishing food (milk, peptonoids, eggs, fruit) and administer stimulants (alcohol, strychnine) if necessary. The leading remedies are *arsenic*, *belladonna*, *carbo veg.*, and *rhus tox.* *Antistreptococcic serum* may be administered.

PYEMIA is a toxemia induced by pyogenic organisms, living bacteria being transported by the blood current to distant tissues where they multiply and produce abscesses (multiple or metastatic abscesses). It is, in short, septicemia plus an embolism which spreads infection to distant parts. Pyemia may be due to surgical operations or injuries, *e. g.*, compound fractures, injuries to veins, etc. It may appear to be "spontaneous" or "idiopathic," usually as the result of osteomyelitis, appendicitis, obscure middle ear disease, etc.

Symptoms.—The typical manifestations of pyemia require ten days or more for their development, but they may be preceded by indefinite septic symptoms, or by actual septicemia. Following this period of incubation, the patient is seized with

a *chill*, which is repeated whenever a fresh suppurative focus is set up. The temperature range is *septic* in type; *i. e.*, fever is intermittent and so irregular that it may be necessary to take the temperature every two hours in order to trace the abrupt changes. *Sweats* accompany the frequent falls in temperature. *Local symptoms* are added to the general signs of suppuration, and vary according to the organ attacked; for example, if the lungs, there will be pain, dyspnea, cough, and expectoration; if the liver, jaundice and symptoms of hepatic abscess; if the heart, ulcerative endocaditis, etc. A very characteristic condition is suppurative arthritis, especially of the sterno-clavicular articulation; it is painless unless complicated with a metastatic osteomyelitis. The mind is usually clear, the patient being restless and hyperesthetic.

Treatment.—That of septicemia.

SCARLET FEVER.

(Scarlatina; Scarlet Rash.)

Etiology.—The *exciting cause*, while probably a micro-organism, remains undiscovered. The disease is communicated by direct contact, by fomites, and by food, the bearer of the contagium being the desquamated epithelium. Children are particularly susceptible. One attack confers immunity as a rule.

Pathology.—The only characteristic lesion is that of the skin, which becomes hyperemic and swollen, the inflammation leading to death and desquamation of the epidermis. The mucous membrane of the throat is erythematous, and secondary changes occur in the cervical glands, the ear, and the heart, kidneys, and other viscera as the result of fever and septic conditions.

Symptoms.—1. *Onset.* The invasion is sudden; there may be chills or chilliness, or in young children convulsions, and *sore throat*, *vomiting* and *high fever* develop quickly. The face is flushed, the eyes dry and dull, and the tongue is coated

with a thin white fur through which the papillæ stand out in distinct red points—the "*strawberry tongue.*"

2. *Fever.* The temperature rises rapidly to 103° or more at the outset, remains high for three or four days until the eruption has appeared, and then falls by lysis, reaching the normal point within two weeks.

3. *General febrile symptoms.* The patient is restless, and complains of headache; respiration is rapid, the bowels are constipated, and the urine is scanty, high-colored and often albuminous. Delirium, coma, and convulsions may ensue; the latter, occurring late in the disease, strongly suggest the possible presence of uremia.

4. *Throat symptoms.* The mucous membrane of the throat is red and there is soreness, with swelling of the tonsils and fauces, often causing dysphagia. An exudate resembling that of follicular tonsillitis or even diphtheria may appear on the tonsils.

5. *Eruption.* The rash appears on the second day and consists of discrete, closely packed red points, with reddening of the skin between them so that the entire surface appears scarlet. It begins about the upper chest and soon covers the whole body. It disappears on pressure, as when the finger nail is drawn across it, but returns in a few seconds. In exceptional cases the eruption may be patchy or may not appear on some parts of the body, and in a few malignant cases it becomes petechial. It begins to fade at once, and desquamation follows; the latter varies, according to the severity of the case, from fine scales to large flakes.

Varieties.—1. *Scarlatina simplex*, the ordinary type just described.

2. *Scarlatina anginosa*, in which throat symptoms are severe, the false membrane resembling that of diphtheria, and local suppuration ensues.

3. *Scarlatina maligna*, in which toxemia is so profound that death may occur before the usual symptoms have fully developed.

4. *Scarlatina hemorrhagica*, in which hematuria, epistaxis, and ecchymoses are prominent.

Complications.—The most common as well as serious complication is acute nephritis, which appears in the third or fourth week, when desquamation is nearly completed. The patient becomes restless, feverish, passes a small quantity of dark urine, and dropsy makes its appearance. (See acute parenchymatous nephritis). Among other common complications are glandular suppurations, otitis media, arthritis, endocarditis, and pneumonia.

Diagnosis.—In mild cases without distinctive eruption, doubt may be removed by the discovery of desquamated scales in the underclothing or by the evidences of nephritis.

	SCARLET FEVER.	MEASLES.	DIPHTHERIA.
Onset.	Sudden, with vomiting.	Gradual, with coryza and photophobia.	Gradual.
Rash.	Scarlet color, seen on upper chest within 36 hours.	Darker patches, seen on face on 4th day.	None typical.
Temperature.	High.	High, but drops on appearance of rash.	Low.
Throat.	Much congested.	Not involved.	Dark red early.
Exudate.	If present, resembles follicular tonsillitis.	None.	Characteristic.
Cervical glands.	Usually involved.	Not involved.	Involved.

Prognosis.—Guarded; the mortality varies from 5 to 30 per cent. in different epidemics.

Treatment.—The patient must be placed at rest in an isolated room, the diet must be liquid, water may be given freely, and tepid sponge baths are advisable. During the early stages, *aconite* is indicated in sthenic cases with great restlessness, *belladonna* in drowsy and dull patients, *gelsemium* in dull, prostated cases, and *veratrum viride* in cases manifesting arterial rather than mental excitement. If the eruption does not appear within 36 hours, place the patient in a hot bath (100°) for ten minutes, and repeat in 3 or 4 hours if necessary.

Upon the appearance of the eruption *rhus tox.* is often indi-

cated. Marked nervous symptoms, with a tendency to coma or convulsions, will require the administration of such remedies as *ailanthus*, *camphor*, *cuprum*, *hydrocyanic acid* or *zinc*. Severe throat symptoms will afford indications for *apis*, *arsenic*, *belladonna*, the *mercurials*, *lachesis*, or *rhus*. Suppurative conditions require such remedies as *arsenic*, *hepar*, *mercury* and *sulphur*. Aural, renal and other complications must receive appropriate treatment.

MEASLES.

(Rubeola; Morbilli.)

Etiology.—The *exciting cause* is an unknown micro-organism. The disease occurs in epidemics, occasionally sporadically, and is usually conveyed by personal contact, rarely by fomites. The contagium is present in the nasal, bronchial and other discharges, as well as in the exfoliated epidermis.

Pathology.—Measles occasions no apparent change in the tissues, the only lesions found post mortem being the result of complications, such as pneumonia and nephritis.

Symptoms.—1. *Catarrhal Symptoms.*—At its onset the disease resembles an ordinary *coryza*, with excoriating discharges from eyes, nose, and throat, photophobia, and dry cough.

2. *Fever.*—The temperature rises moderately at the outset (102–104°), but on the second day there is a decided remission which lasts until the eruption appears on the fourth day; then it rises rapidly, but falls again as soon as the rash is established.

3. *Koplik's Sign.*—On the second or third day minute bluish white specks surrounded by a red areola appear on the mucous membranes of the cheeks and lips.

4. The *eruption* appears on the palate about the third day in the form of numerous red points and blotches. On the fourth day the rash appears on the skin, first on the face and then over the entire body. At first it is red and punctiform, but in a few hours the small rounded red spots become slightly

elevated, and as they enlarge form patches which are often crescentic in form and, as a rule, disappear on pressure. At the end of two or three days the rash fades gradually and fine desquamation ensues.

"Black" measles is a malignant type of the disease characterized by subcutaneous extravasations of blood, hemorrhages from the mucous surfaces, and profound prostration which often leads to death.

Complications. — Ophthalmia, otitis, stomatitis, laryngitis, broncho-pneumonia, and nephritis.

Diagnosis.—

Eruption.

	Occurrence.	*Character.*	*Duration.*	*Location.*	*Desquamation.*	*Duration of Disease.*
Measles.	4th day.	Small red macules, crescentic borders.	4 to 5 days.	Face, then body.	Branny.	2 weeks.
Rubella.	Within 48 hours.	Round red discrete macules.	3 days.	Face and scalp, then body.	Slight, branny.	4 to 7 days.
Scarlatina.	Within 24 hours.	Diffuse, red, punctate.	7 to 10 days.	Neck and Chest, then face and body.	Scales.	2 to 3 weeks.
Variola.	4th day.	Umbilicated pustules.	21 to 25 days.	Face, then body.	Crusts.	4 to 5 weeks.
Varicella.	Within 12 to 24 hours.	Crops of vesicles.	5 to 8 days.	Back, chest, arms.	Crusts.	2 weeks.
Erysipelas.	Within 24 hours.	Bright red, polished, with raised margins.	4 to 8 days.	Face.	Branny.	1 to 3 weeks.

Prognosis.—In healthy children with good surroundings the prognosis is generally favorable. Complications may render it grave.

Treatment.—Place the child at rest in a large, dark, well-ventilated room, restrict the diet to milk, soups, etc., and continue these measures until the temperature has been

normal for a week. The patient should be bathed daily in tepid water, and the eyes and mouth cleansed with a simple antiseptic solution, such as boric acid, 10 grains to an ounce of distilled water.

The early febrile symptoms will suggest the use of *aconite*, *veratrum viride*, or *belladonna*. The catarrhal symptoms, if prominent, will require such medicines as *euphrasia*, *kali bichrom.* or *pulsatilla*. If the bronchial tubes are severely attacked, a dry cough will require *bryonia*, *drosera*, *sticta* or *lachesis;* and free secretion will indicate the need of *antim. iod.*, *arsen.* or *tartrate*.

RUBELLA.

(Rotheln; German Measles.)

Etiology.—The *exciting cause* of this mild, though highly contagious, disease is an unknown micro-organism. It occurs in epidemics, rarely sporadically, and generally affects children.

Symptoms.—Without appreciable prodromes, the patient becomes mildly feverish (100°), and may complain of chilliness or headache. On the first or second day an indistinct macular eruption appears and develops into pale-red papules, which may remain discrete like measles (*rubella morbilliforme*) or may become small, bright red and diffuse, resembling scarlet fever (*rubella scarlatiniforme*). Glandular enlargements can be detected about the neck, and the patient complains of sore throat. The eruption may spread over the entire body, but it fades rapidly and in the course of a week it and all other symptoms have disappeared.

Prognosis.—Good. Complications are almost unknown.

Treatment.—Rest, light diet, and such remedies as *aconite*, *belladonna*, *pulsatilla* and *rhus*.

VARIOLA.

(Smallpox.)

Etiology.—The disease is highly contagious, the virus being thrown off by the lungs and skin, and few of those unprotected by vaccination escape infection if exposed freely. The poison may remain latent in fomites for years. The specific organism has not yet been recognized.

Pathology.—The essential lesion is an inflammatory cellular

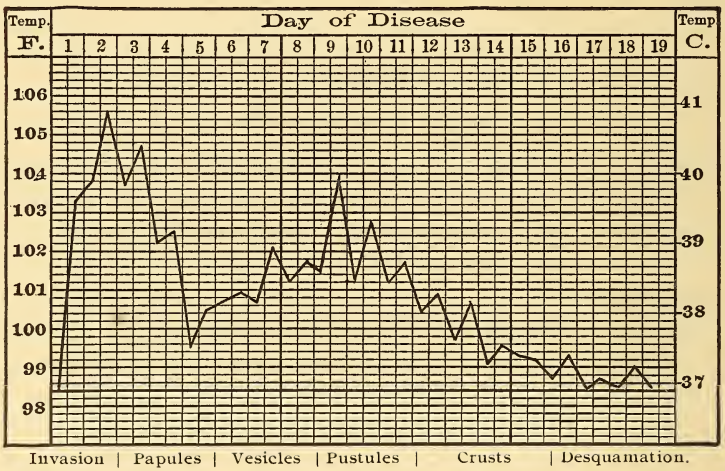
Temp. F.
Day of Disease
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19
Temp C.
106 105 104 103 102 101 100 99 98
41 40 39 38 37
Invasion | Papules | Vesicles | Pustules | Crusts | Desquamation.

FIG. 7. Chart showing the Temperature Curve and Course of the Eruption in Small-pox (Salinger-Kalteyer).

infiltration of the *rete mucosum*, accompanied by profound toxemia and the consequent degenerative changes in the viscera.

Clinical Course.—The onset is sudden, a violent *chill* being followed by a *rise of temperature* to 103° to 104° in the first twenty-four hours, with a more gradual but continued rise for the next three days. At this time headache and *backache* are very severe, and may be accompanied by pains in the limbs, nausea, vomiting and photophobia.

The *eruption* appears especially on exposed portions of the body about the fourth day. At first it consists of small red macules, which rapidly change to papules and give a "*shotty feel*" to the skin. About the sixth day these papules become converted into clear *vesicles with umbilicated centres*. By the eighth or ninth day of the disease the vesicle becomes a pustule and umbilication disappears. At this time the skin between the lesions becomes swollen and edematous, and the patient's features are hideously unrecognizable. In about three days more the pustules begin to dry up and form offensive smelling crusts which, falling off, leave permanent scars or "pock-marks." Frequently the eruption involves not only the whole surface of the body but the mucous membranes of the respiratory and gastro-intestinal tracts.

With the appearance of the eruption the temperature falls suddenly, but not quite to normal, and the constitutional symptoms lessen greatly. When the pustular stage is reached, however, a secondary or suppurative fever occurs; the temperature rises again and the severe symptoms return. As dessication occurs the fever falls gradually until finally convalescence begins.

Varieties.—I. *Variola vera*, which is divided into two forms: *Variola discreta*, in which the lesions are isolated; and *variola confluens*, in which the pustules become confluent.

II. *Variola hemorrhagica* (black smallpox), which is divided into *purpura variolosa*, in which blood appears in the papules in the first days; and *variola hemorrhagica pustulosa*, in which blood appears in the vesicle or pustule, at times in association with hemorrhages from mucous surfaces.

III. *Varioloid*, a modified smallpox, mild in its course, which attacks those protected by a previous attack or by vaccination.

Complications.—Inflammations of the eye and ear; bronchitis; catarrhal pneumonia; abscesses and localized gangrene.

Diagnosis.—Prior to the appearance of the eruption, diagnosis is difficult, although the sudden high fever with intense head-

ache and backache is significant, and the "shotty feel" beneath the skin is yet more so. *Chicken pox* presents a similar eruption, but the latter appears in successive crops and is not accompanied by high fever.

Prognosis.—The mortality varies with the severity of the epidemic and the previous condition of the patient; it is rarely below 20 per cent. Confluent and hemorrhagic cases are extremely fatal.

Treatment.—Promptly isolate the patient in a well-aired room, and restrict his diet to liquids. The immediate vaccination of every individual exposed to infection is imperative.

Local antiseptic treatment, designed to prevent pitting, may be instituted; it consists in covering the exposed portions of the body with cloths soaked in a solution of mercuric chloride, 1–10000. When pus forms, evacuate with a fine needle and apply carbolized dressings.

In the stage of invasion the remedies indicated may be *aconite*, *belladonna*, *gelsemium* or *veratrum viride*. In the stage of eruption indications are often present for the administration of *rhus tox.*, *apis mel.*, *baptisia*, *croton tig.*, *sulphur*, *tartar emetic* or *thuja*. *Vaccine virus* has been given internally with benefit to the patient. *Hepar* has been employed to limit the suppuration. Hemorrhagic cases require such remedies as *lachesis*, *crotalus*, *hamamelis*, *phosphorus*, *hydrochloric acid* and *arsenic*.

VACCINATION.

(Vaccinia.)

The inoculation of human beings with the virus of cowpox was introduced by Jenner in 1798, and to its efficacy in the prevention of smallpox must be attributed the virtual disappearance of that frightful pest.

The method of vaccination is extremely important. Lymph taken from a healthy cow and freed of impurities by preservation in glycerine, should be used. The spot selected for the introduction of the virus is usually over the insertion of the

deltoid in the left arm, or on the outer aspect of the thigh immediately above the knee. After careful cleansing, a small area of skin is denuded superficially with an ordinary scalpel until red serum begins to ooze. The vaccine virus is then rubbed in, and the part is exposed to the air until thoroughly dry and is afterwards covered with a shield.

The course of a primary vaccination should be typical. In a normal case a papule appears on the second or third day and about the fifth day this becomes an umbilicated vesicle surrounded by an inflammatory zone. About the eighth day the vesicle changes to a pustule, and by the twelfth day a crust has formed, which subsequently drops off and leaves a scar.

Every child should be vaccinated, preferably between the second and third months of life, and thereafter every seven years. Occasionally hot weather or ill health may justify a postponememt. The presence of an epidemic of smallpox demands immediate vaccination or re-vaccination without regard to circumstances.

Complications of vaccination, such as ulceration, erysipelas, or septicemia, are positive evidence of fault on the part of the virus or the vaccinator. Fortunately they are extremely rare. Should they occur, the treatment must be conducted on general principles.

VARICELLA.

(Chicken-pox.)

Etiology.—This is a mild infection, usually occurring in childhood; it is contagious, but the specific cause has not been determined.

Symptoms.—Small red spots appear on the skin and mucous membranes and develop rapidly into vesicles, which in a day or so dry up into crusts and fall off, leaving no scar. Fresh *crops* of eruption appear on each of the first few days, so that all stages are present at one time. Rarely it becomes pustular and leaves a scar. The attack is accompanied by febrile symptoms and a slight rise of temperature.

Diagnosis.—The lesion is superficial, rarely umbilicated, and lacks the red areola seen in smallpox; it appears in crops and involves the surface of the trunk to a greater extent than does variola.

Prognosis.—Absolutely good. Rarely acute nephritis has occurred as a sequel to the attack.

Treatment.—Isolation, rest and light diet, together with the administration of *rhus tox.*, *mercurius*, *vaccininum* or *tartar emetic.*

WHOOPING COUGH.

(Pertussis.)

Etiology.—The disease is probably due to a specific organism whose toxin has a selective action on the centers of the vagus and superior laryngeal nerves. As a rule it occurs in children, who acquire it by contact, and it is presumed that the virus is conveyed in the sputum.

Pathology.—The only lesions discovered post-mortem are those due to complications, such as bronchitis, broncho-pneumonia and pulmonary collapse.

Symptoms.—1. *Catarrhal stage.* The disease begins like an ordinary cold, with lachrymation, coryza, dry cough, and feverishness. In the course of ten days this merges into the

2. *Paroxysmal stage.* The cough becomes violent and occurs in paroxysms so prolonged that the patient becomes cyanosed and seems in danger of suffocation; then a long, crowing inspiration—the "*whoop*"—takes place, a quantity of glairy mucus is expectorated or vomited up, cyanosis disappears, and the paroxysm is over. This may be repeated anywhere from four to fifty or more times in twenty-four hours, the severity of the disease being judged by the frequency of the attacks. After from three to six weeks the disease passes into the

3. *Stage of decline*, in which paroxysms become less frequent and less violent and finally cease altogether. The

entire course of the disease extends over ten or twelve weeks.

Complications.—The violent coughing may cause hemorrhagic symptoms: such as petechiæ in various parts of the body, epistaxis, hemoptysis, or even sub-dural hemorrhage. Bronchitis, broncho-pneumonia, pulmonary collapse, pleurisy, and interstitial emphysema may occur.

Diagnosis.—By the occurrence of a "whoop."

Prognosis.—Favorable in the absence of complications. The younger the child the greater the danger of the occurrence of the latter.

Treatment.—1. *Prophylaxis.* Children, especially the young and delicate, must be kept from communication with the patient as long as the cough lasts.

2. *General care.* Daily exercise in the open air, abundant and nutritious food, woolen underclothing, and the avoidance of exposure to cold and damp, are essential hygienic details.

3. *Medicines.* In the catarrhal stage, *aconite*, *belladonna*, *hyoscyamus*, or *ipecac* may be indicated. *Naphthalin* may be given early and persistently, or with the advent of the paroxysms the symptoms may call for *coccus cacti*, *corallium rubrum*, *cuprum acet.*, *drosera*, or *mephitis*. In severe cases with menacing symptoms, spasm of the glottis can be absolutely relieved by intubation.

MUMPS.

(Epidemic Parotitis.)

Etiology.—The disease appears in epidemics, is sometimes sporadic, attacks children and adolescents by preference, and one attack confers immunity. The specific germ is undiscovered.

Pathology.—Inflammatory infiltration of the salivary glands.

Symptoms.—1. *Pain below and in front of the ear*, with distress on swallowing.

2. *Enlargement of the salivary glands* about the ear, some-

times accompanied by great swelling of the neck and cheek. In two or three days the other side is involved.

3. *Moderate pyrexia* (101°) accompanies the invasion.

4. *Involvement of the sexual organs* is a peculiar complication of mumps; orchitis in males and inflammation of mammæ or ovaries in women may follow the decline of the parotid swelling.

Diagnosis.—*Secondary parotitis*, occurring as a complication of such acute diseases as typhoid and typhus, is much more serious than mumps on account of its liability to suppuration; it can easily be distinguished by the history of associated disease.

Enlargements of the cervical lymphatic glands, such as occur in scrofulous children, are persistent, while the swelling due to mumps disappears in about ten days.

Prognosis.—Favorable, recovery ensuing in the course of a week.

Treatment.—Rest, and the administration of such medicines as *aconite*, *belladonna*, *mercury*, *rhus*, and *sulphur*. A complicating orchitis requires support and perhaps strapping of the testicles, and *pulsatilla* or *clematis* internally.

TETANUS.

(Trismus; Lockjaw.)

Etiology.—The *exciting cause* is the tetanus bacillus, a long, slender rod with a spore at one end which makes it resemble a drum-stick. This organism is widely distributed in the soil, in rubbish, etc., but it is anærobic, and hence man is rarely infected. Its mode of entrance is through some wound.

Pathology.—The virus acts upon the nerve centres of the medulla and cord, and these, together with the nerve trunks leading from the site of infection, become inflamed.

Symptoms.—At the onset the patient complains of languor and of stiffness of certain muscles, usually those of mastica-

tion; and in a short time tonic spasm of the latter locks the jaws (*trismus*). The cervical muscles also become rigid, retracting the head; the face becomes fixed in a sardonic grin, and the muscles of the trunk and of the spinal column become rigid, and by their retraction arch the body (*opisthotonos*, *pleurosthotonos*, or *emprosthotonos*). At intervals, often from very slight external irritation, convulsions occur and entail frightful suffering. The temperature is raised (103–106°), the pulse is quickened, the body is bathed in perspiration, the bowels are constipated, and the urine may be suppressed. The mind remains clear to the end.

Course.—The disease may be acute, violent, and rapidly fatal; or it may be sub-acute or chronic, and milder in its manifestations, with a tendency to recovery.

Diagnosis.—*Strychnine-poisoning* immediately follows the ingestion of that drug; it begins with gastric disturbance or contraction of the extremities, rather than of the muscles of mastication; violent convulsions alternate with periods of complete relaxation; and examination of the gastric contents reveals the presence of strychnia.

Hydrophobia is distinguished by a history of an animal's bite, by laryngeal rather than masticatory spasm, by psychical disturbances and by the absence of opisthotonos.

Tetany is marked by long-continued spasm of the extremities, particularly the hands, and lock-jaw occurs late or not at all.

Prognosis.—Grave, the mortality approximating 80 per cent.

Treatment.—Disinfect and cauterize the wound with nitrate of silver; re-open it if necessary. Place the patient at absolute rest in a room from which light and sound are excluded. Feed per rectum. *Chloral hydrate*, gr. xx-xxx, may be given by mouth or rectum to control the spasms; or it may be necessary to resort to *chloroform* inhalations. *Strychnine* is the leading internal remedy; *aconite*, *gelsemium*, *hydrocyanic acid*, *passiflora*, and *tetanus antitoxin* may also be considered.

ANTHRAX.

(Malignant Pustule; Splenic Fever; Wool-sorter's Disease.)

Etiology.—The *exciting cause* is the anthrax bacillus, an organism which especially attacks herbivorous animals; the latter derive it from the soil, not from each other. Occasionally it gains access to the human body through wounds, the bites of insects, or with infected food or contaminated air. Those associated with cattle or their products, such as hides or hair, are necessarily exposed to infection.

Pathology.—At the point of infection, whether external or internal, local inflammatory changes develop rapidly and eventuate in gangrene rather than suppuration. The gangrene is due to the mechanical effect produced by the enormous multiplication of the organisms, which through the blood are also enabled to invade every organ. The spleen in particular is greatly enlarged, and all the viscera are degenerated.

Symptoms.—*External anthrax.* At the point of inoculation, usually on an exposed portion of the body, a pimple appears, enlarges, and quickly becomes a vesicle filled with blood. This bursts, leaving a raw, necrotic surface; and the latter is surrounded with a dusky ring of induration, which may be set with vesicles. In a few hours severe constitutional symptoms set in; there is prostration, fever, and delirium, and death may soon occur; but if the slough be thrown off recovery is possible.

Internal anthrax may involve the lungs (wool-sorter's disease) or the intestines (intestinal mycosis). *Wool-sorter's disease* is characterized by symptoms of bronchitis, with dyspnea, pain in the back and legs, and profound prostration, which soon ends in death. *Intestinal mycosis* presents the symptoms of acute poisoning, with chill, abdominal pain, nausea, vomiting, intense prostration, and death in a few days.

Diagnosis.—External anthrax must be distinguished from ordinary *carbuncle.* The latter appears oftener upon the back

of the neck or upon covered portions of the body, pursues a less rapid course, is more painful than anthrax, and presents multiple openings ("pepper-box lid").

Internal anthrax cannot be recognized by its symptoms alone. Knowledge of the patient's exposure should lead to a search for the bacillus in the blood; it stains readily with Lœffler's methylene blue solution.

Prognosis.—External anthrax, being simply a local infection at the outset, can be cured in a majority of cases by early cauterization of the lesion, but if unrecognized or untreated, the developing toxemia renders it almost certainly fatal. Recovery from internal anthrax is exceptional.

Treatment.—Prophylaxis should be insured by the cremation of the bodies of diseased animals. As soon as the external lesion is recognized in man, it should be excised, and its site cauterized with the hot iron or acids.

RABIES.

(Hydrophobia; Lyssa.)

Etiology.—The exciting cause, while certainly a micro-organism, is unknown. Rabies is a disease of the lower animals, especially the carnivora, by whose bite it is transferred to man.

Pathology.—The disease is marked by an inflammatory infiltration in the medulla, especially about the respiratory centre, congestion of the cord, and hyperemia of the respiratory mucous membrane.

Symptoms.—1. *Premonitory stage.* Following the inoculation, usually by a dog's bite, there is a prolonged period of incubation (2–6 months), and during this period the patient may be depressed and irritable, and the wound, after completely healing, may show evidences of irritation.

2. The *stage of excitement* follows. The patient's face betrays intense anxiety, and he becomes so remarkably hyper-

esthetic that a breath of air or a mere touch may induce a violent spasm of the larynx. Through fear of inducing this paroxysm he dreads even the sight of water, though he is thirsty. His excitement may become maniacal, or repeated spasms of the larynx may cause dyspnea, and so lead to the emission of curious sounds. During the intervals between the paroxysms the patient's mind is perfectly clear. There is usually moderate fever, with a rapid pulse and quickened respiration.

3. The *paralytic stage* supervenes in from one to three days; the spasms subside, the patient becomes quiet, general paralysis gradually develops, and in the course of a few hours he becomes unconscious and dies.

Diagnosis.—*Hysteria* may develop in those who have been bitten by dogs. Such patients try to bark like a dog and to bite themselves and others; pulse and respiration are unaffected, and by the history and the stigmata the real nature of the disease can be determined.

Prognosis.—When the disease has become established the prognosis is hopeless.

Treatment.—Prophylaxis should be secured by the cauterization of all wounds received in the form of bites, and by preventive inoculations with the emulsion made with the cord of a diseased animal (Pasteur). During the attack *chloral* or *morphine* may be given internally, and *chloroform* inhalations may be used in order to lessen the suffering. *Belladonna* or *cantharis* may be administered on symptomatic grounds.

GLANDERS.

(Farcy.)

Etiology.—The *exciting cause* is the bacillus mallei, an organism which is a little shorter and thicker than the tubercle bacillus. The disease is one of horses, but is occasionally transferred to man by the inoculation of some small wound with the nasal discharge from the animal. In consequence

those whose occupation brings them in contact with horses are alone liable to the infection.

Pathology.—At the point of inoculation (usually on the nasal mucous membrane or skin) a nodule, composed of round cells, develops, and this soon breaks down and forms an ulcer. The infection spreads by way of the lymphatics, and ultimately the lesions may appear throughout the viscera as well as under the skin.

Symptoms.—At the point of infection a nodular swelling, which may vary in size from a pea to a walnut, appears, softens, and breaks down to form a spreading ulcer which discharges fetid pus (*farcy*) If the nasal cavity be the point infected, the mucous membrane becomes swollen and studded with nodules which subsequently ulcerate; and the destructive process may involve the nasal septum, mouth, pharynx, and even the lower respiratory tract (*glanders*).

In either form of the disease the local symptoms are accompanied by intense prostration, muscular pains, high temperature, emaciation, and finally the typhoid state and death. The disease may be *acute*, terminating within three weeks, or *chronic*, extending over months.

Diagnosis.—By the appearance of characteristic nodules, in the discharge from which the organism is found. For its detection, stain a cover glass preparation with warm carbolfuchsin, and decolorize in a 2% solution of nitric acid.

Prognosis.—In acute cases, unfavorable; in cases of more chronic course, about 50% of the patients recover.

Treatment.—Surgical treatment (cauterization) of the primary lesion, with generous nourishment and if necessary stimulation of the patient. Internally, *kali bich.*, *arsenic*, or *graphites* may be indicated.

ACTINOMYCOSIS.

(" Big-jaw;" " Lumpy Jaw," Etc.).

Etiology.—This disease, which affects both men and cattle, is caused by the actinomyces or ray-fungus. The latter gains entrance through the mouth, teeth, or pharynx, being introduced with food or drink; rarely it obtains entry through the respiratory tract or skin.

Pathology —By the growth of the fungus macroscopic yellow masses are produced, which vary in size from a millet-seed to an orange. The tissues surrounding the tumor become inflamed, and ultimately, as the result of secondary pyogenic infection, suppuration occurs. Microscopically the granular masses are seen to consist of a closely woven group of conical threads with their club-shaped ends outward.

Symptoms.—Clinically these masses are found in the mouth, the respiratory tract, and the alimentary canal. The growth of the tumor is slow, causing little pain, but finally it suppurates and discharges a fluid containing characteristic yellow granules. If it occurs upon the jaw, the bone becomes enlarged and the neck changed in shape. If the lesion is pulmonary, the symptoms resemble broncho-pneumonia or tuberculosis. The intestinal variety presents the symptoms of gastro-enteritis.

Diagnosis.—Whatever the localization of the lesion, the recognition of its nature rests upon the discovery of the fungus in the discharges.

Prognosis.—When the lesion is accessible to surgical interference, cure is possible; if not, the patient usually succumbs with symptoms of pyemia.

Treatment.—Surgical interference if possible; if not, *potassium iodide* in large doses.

TUBERCULOSIS.

Etiology.—The *exciting cause* is the tubercle bacillus, a slender rod less than half the diameter of a red blood corpuscle in length, which gains entrance to the body—

1. By *inhalation*, the dust of dried sputum being the ordinary infective medium.

2. By the *ingestion* of infected food, such as the meat or milk of tuberculous cattle.

3. By *inoculation*, as in the cases of pathologists and others working with infected tissues.

4. By *direct parental transmission* through the placenta (rare).

Among *predisposing causes* are:

a. Tuberculous heredity.

b. Loss of resisting power, as the result of insanitary living, frequent child-bearing and prolonged lactation, occupations amid unhealthful surroundings, or exposure to dust-inhalation, and certain previous diseases, such as pneumonia, bronchitis, diabetes, etc.

c. Climatic conditions, such as warmth and moisture associated with defective drainage.

d. Age. No age is exempt, but a large proportion of cases occur in early life, the lymphatic glands and bones in children, and the lungs in young adults, being especially liable to the infection.

General Pathology of Tuberculosis.—The tubercle bacillus, reaching the tissue through the inspired air, the lymph, or the blood, or with ingested food, produces local irritation at the point of its lodgment; and the result is:

1. The *formation of a small grayish granular body*, perhaps the size of a millet-seed—a *tubercle.* The steps in its formation are as follows:

(*a*) Proliferation of the fixed cells of the tissue upon which

the bacillus has lodged, the new cells being of large size and containing a single oval nucleus—the so-called epitheliod cells.

(*b*) The formation of a wall of leucocytes around this group of new cells.

(*c*) The appearance in the midst of the new structure of a few very large cells, consisting of coarsely granular protoplasm with many nuclei—giant cells.

(*d*) Degeneration in the centre of this nodule, which is cut off from the blood supply and poisoned with bacterial toxins; and this necrotic process soon transforms the central portion into a yellowish, cheesy mass.

Histologically, the tubercle appears to consist of an outer layer of small round cells (leucocytes), a middle zone of epitheloid and a few giant cells, and a caseous centre. A fine fibrillary reticulum appears at the margin, the fibrils lying between the epitheloid cells; and the tubercle bacillus is present in and between the cells in large numbers. A tubercle may coalesce with neighboring nodules either before or after it undergoes caseation.

2. *Diffuse inflammation of the tissues about the tubercle*, which may become obscured by the rapid filling of the tissues with cell-products and inflammatory exudate. The latter quickly becomes caseous, and results in a diffuse cheesy infiltration, as seen, *e. g.*, in brain, kidneys, testicles, etc.

The *caseous mass* thus formed may undergo:

(1) *Softening.* Septic organisms, especially streptococci, may gain entrance to the nodule, setting up suppuration; and ultimately the pus may be discharged, leaving behind a cavity with ulcerating walls. Or,

(2) *Fibrosis.* An overgrowth of connective tissue may spring up around the nodule, completely encapsulating it; and as a result the caseous mass may remain unchanged for years, or by deposit of lime salts it may become *calcified.* Tubercular lesions never undergo resolution.

ACUTE TUBERCULOSIS.

(Acute Miliary or General Tuberculosis.)

Etiology.—By the breaking down of a caseous focus in some part of the body—most frequently in lung or lymphatic gland—tubercle bacilli are thrown into the blood current and distributed throughout the body. Tuberculous lesions are thus produced in many organs. Clinically, three distinct forms are recognizable, one presenting symptoms of general infection without distinct localization, a second presenting predominant

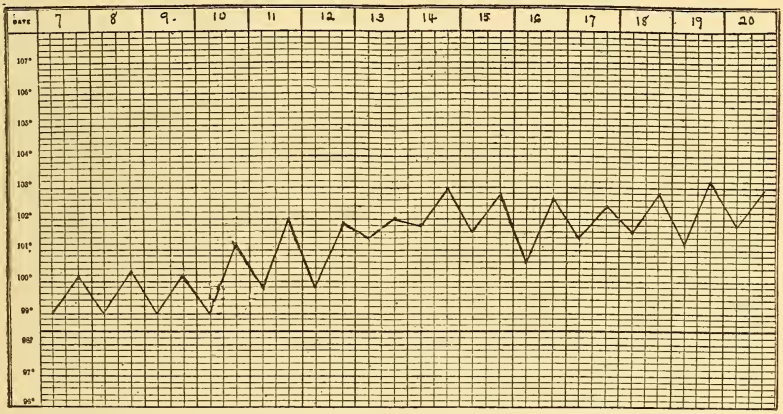

FIG. 8.—Chart showing the Irregular Continued Fever of Miliary Tuberculosis.

pulmonary symptoms, and a third of cerebro-spinal type.

Symptoms.—I. TYPHOID FORM. After some days or weeks of ill-defined prodromes, there appear—

1. *General Febrile Symptoms.*—Headache, mental dulness and ultimately delirium and coma occur. The tongue becomes dry and brown, there is rapid loss of flesh and strength, and sometimes diarrhea or intestinal hemorrhage is associated. The occurrence of excessive sweating accompanied by sudamina, and the presence of herpes may serve to exclude typhoid fever.

2. *Symptoms due to certain localizations* may occur, *e. g.*, cough, usually dry, with hurried respiration, dyspnea and cyanosis; pleural friction; pericardial friction; tubercles in the choroid coat of the eye.

3. *Pyrexia.* — The temperature is high (103°–107°), but irregularly and markedly intermittent; occasionally there are characteristic "inversions" in which the maximum rise occurs in the morning instead of the evening. Pulse and respiration are rapid.

Diagnosis.—

Acute miliary tuberculosis.	*Typhoid fever.*
Epistaxis rare.	Epistaxis common.
Rash unusual.	Characteristic rose-spots on abdomen.
Respiration hurried, dyspnea, cyanosis.	Respiration regular.
Rales moist, subcrepitant.	Rales dry, sonorous, sibilant.
Temperature irregular, may be intermittent or inverted.	Temperature shows regular diurnal remission.
Tubercles may be found on choroid.	None.
No Widal reaction.	Widal reaction present.

Prognosis.—Invariably fatal. Toward its termination the disease may assume the pulmonary or meningeal type.

II. PULMONARY FORM (miliary tuberculosis of the lungs). Gradually, sometimes suddenly, the patient develops:

1. *General Febrile Symptoms.*—At first there is headache, malaise, and loss of appetite; delirium and coma may come later. An initial chill is rare.

2. *Pulmonary Symptoms.*—These vary. *Hemoptysis* may occur at the outset if there is an old tubercular lesion in the lung. *Cough* is usually present, but is not always severe. *Expectoration* may be absent; if present it is mucoid and may be blood-stained. Pleuritic pain is sometimes noted. *Dyspnea* begins early, and as the disease advances the respirations may become very rapid (50–80 per minute). *Cyanosis* appears when considerable lung structure is involved and increases to the end.

3. *Pyrexia* may be slight if debility is extreme; but, as a

rule, the pulmonary form is characterized by continuous fever (101°–104°) with slight morning remissions. Less often it is of the inverse type, attaining its maximum in the morning instead of the evening.

The pulse is rapid and feeble, the spleen enlarged, albuminuria is common, and the typhoid state may develop.

Diagnosis.—The physical signs are not distinctive, being those of broncho-pneumonia. In doubtful cases search should be made for old tubercular foci, for tubercles in the choroid, and for bacilli in the sputum. The onset of the symptoms of tubercular meningitis will also confirm the diagnosis.

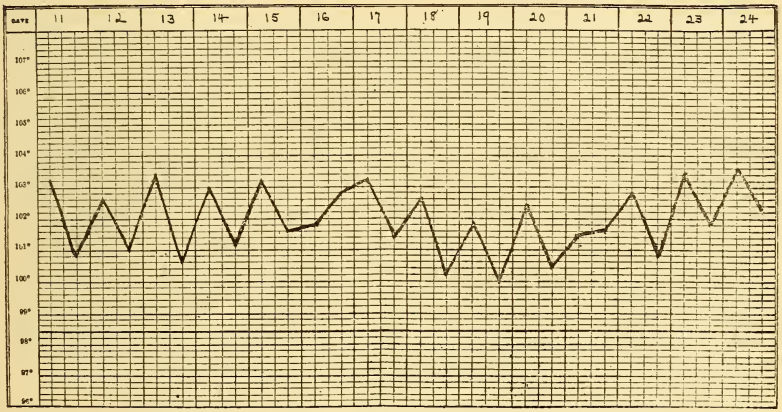

FIG. 9.—Chart showing the "Inverted" type of Fever in Miliary Tuberculosis.

III. MENINGEAL FORM (tubercular meningitis; basilar meningitis; acute hydrocephalus). In this form there is a deposition of tubercles along the vessels of the pia mater at the base of the brain, with an exudation of lymph which renders the membranes thick and opaque. For some weeks the patient, usually a child, appears indefinitely ill; then he develops—

1. *Intense headache and delirium*, with hyperesthesia of the special senses. The pain may be excruciating, causing the patient to utter penetrating screams (hydrocephalic cry). The pupils may be contracted.

2. *Vomiting of cerebral type*, without nausea and without great effort.

3. *Convulsions, paralysis, and coma.* According to the localization of the lesion, twitchings of various groups of muscles may occur, to be followed later by paralysis. Thus there may be produced strabismus, ptosis, or paralysis of face or limbs. General convulsions may occur.

4. *Respiration* becomes slow, irregular and sighing as the disease progresses.

5. *Pyrexia* develops slowly and is rarely high (102°–103°). The pulse is either slow or but moderately accelerated. The typhoid state may develop.

Prognosis. — Unfavorable, the disease usually terminating fatally within four weeks.

Treatment.—In a disease so hopeless, whichever form it may assume, little else can be done than maintain the strength of the patient by means of rest and nutritious food. Among remedies symptomatically suitable are *arsenic*, the *calcareas*, *cinchona*, *ferrum*, *hyoscyamus*, *iodine*, *phosphorus*, *rhus tox.*, *stramonium*, and *sulphur*. In the meningeal cases *apis*, *belladonna*, *bryonia*, and *helleborus* are also recommended. A few cures have been reported as following the repeated application of iodoform ointment to the shaved head.

TUBERCULOSIS OF THE LYMPHATIC GLANDS.

(Scrofula; Tuberculous Lymphadenitis.)

Involvement of the lymphatic glands, especially the cervical, bronchial, and mesenteric, is common; they become swollen, and in many cases suppurate and finally rupture.

Cervical lymphadenitis is obvious on inspection or palpation of the neck; it is necessary, however, to exclude Hodgkins' disease, leucocythemia, and sarcoma.

Bronchial lymphadenitis may be unaccompanied by symptoms, or on the other hand the enlargement may be so great as to

suggest mediastinal tumor. Symptoms may arise from pressure upon the pneumogastric or recurrent laryngeal nerves (spasmodic cough, dyspnea, hoarseness, spasm or paralysis of laryngeal muscles, acceleration or slowing of the heart) or from rupture of a softened gland into a bronchus (cough with expectoration of pus, blood and bacilli).

Mesenteric lymphadenitis (tabes mesenterica) may be accompanied by symptoms of associated peritonitis, diarrhea, fever, painful and swollen abdomen, and evidences of peritoneal effusion. Detection of the nodules, by careful palpation, will establish the diagnosis.

PULMONARY TUBERCULOSIS.

(Phthisis Pulmonalis; Consumption of the Lungs.)

Pathology.—Deposit of the tubercle bacillus in the lungs is followed by the formation of—

1. *Tubercles.*

2. *Diffuse lesions*, including—

(*a*) True pneumonic inflammation of the broncho-pneumonic type.

(*b*) Proliferation of the connective tissue.

(*c*) Inflammation and adhesion of the pleura over the affected lung area.

The areas involved may undergo caseation, destruction, and excavation, or they may undergo fibrosis or calcification.

Varieties.—The forms of pulmonary tuberculosis differ from each other simply in the predominance of caseation and softening or of fibrosis in the lesion; they are as follows:

I. *Miliary tuberculosis of the lungs.* This variety is marked by a rapid formation of miliary tubercles throughout the lungs. It may be simply a terminal event in chronic tuberculosis, it may follow the break-down of an old encapsulated lesion, or it may be primary. It is often associated with the formation of miliary tubercles in many other organs, to which the bacilli

have been conveyed by the blood. (See General Tuberculosis, page 89).

II. *Caseous tuberculosis of the lungs* (acute pneumonic phthisis, florid phthisis, galloping consumption) occurs in persons whose resistance is so weak that nearly all the lung structure in one area after another is involved in the tubercular process, undergoing rapid caseation and softening. This variety may occur in a *lobar* form, in which there is a massive consolidation, a lobe or even an entire lung becoming caseous; or, more often, a *disseminated* form, in which the process begins in the bronchioles of one or both lnngs, quickly spreads to the alveoli, and is distinctly broncho-pneumonic in type.

III. *Fibroid tuberculosis of the lungs* represents an extreme opposite to the variety just described. The miliary tubercles undergo fibrosis before either caseation or softening can occur, appearing in the lung as:

(*a*) Isolated granulations, indurated and deeply pigmented;

(*b*) Groups of such granulations;

(*c*) Rounded or irregular areas of induration surrounded by fibrous granulations.

IV. *Fibro-caseous tuberculosis of the lungs* (chronic, ulcerative tuberculosis), the common form of the disease, stands midway between the purely caseous and the fibroid forms. Areas of caseation are formed and often undergo softening and excavation, but inflammatory changes at the margin of the lesion lead to the production of a fibrous wall which checks the advance of the disease for a time. At intervals fresh areas are attacked by the infection and occasion active symptoms; then the process becomes checked again, and another period of apparent latency occurs. In this way the disease may extend over months and years.

CASEOUS TUBERCULOSIS OF THE LUNGS.

(Acute Pneumonic Phthisis; Florid Phthisis; Galloping Consumption.)

Symptoms.—1. *Onset.* As a rule the initial symptoms and physical signs are those of any *pneumonic* process, the onset being attended with fever and cough. Sometimes there is an initial chill, and occasionally hemoptysis is the first symptom.

2. *Hectic symptoms.* In a short time the fever assumes the hectic type, with high temperature, flushed cheeks, hot skin,

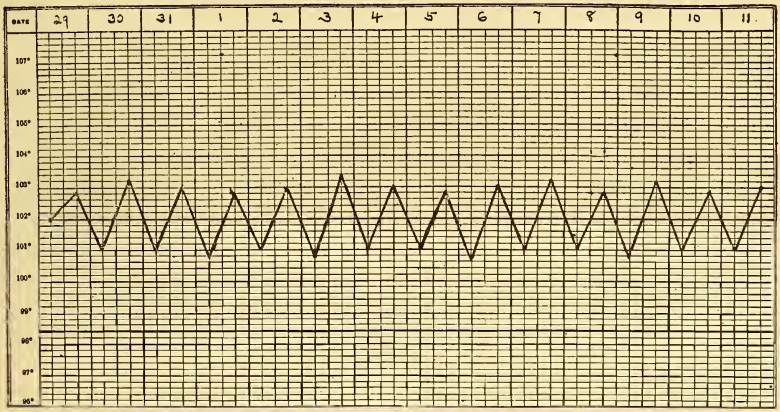

FIG. 10.—Chart showing the High Remittent Fever of Caseous Tuberculosis.

night sweats, chills, and rapid emaciation.

3. The *expectoration*, at first mucoid and perhaps blood-stained, becomes purulent and contains tubercle bacilli.

4. *Pyrexia.* The temperature at first is high (101–104°) with marked remissions; later, with softening and excavation in progress and vitality failing, it may become intermittent, with an evening rise to 102° or more, and a morning fall to 97° or below.

Physical Signs.—In the *broncho-pneumonic* variety the signs are those of infiltration, viz., lessened resonance, harsh breath-

ing, and crepitation, usually at the apices; and these may merge into the signs of consolidation, including dulness, bronchial breathing, and bronchophony. In the *lobar* form the symptoms are those typical of pneumonia, viz., percussion-dulness, crepitation, and tubular breathing over an extensive area. In either form signs of excavation may be detected later.

Diagnosis.—Even though the nature of the infection be unsuspected at the outset, the absence of a crisis at the end of a week or so, and the rapid pulse, sweating, and unusual emaciation, will suggest examination of the sputum; and discovery of the tubercle bacilli, often in association with fibres of connective tissue, will reveal the nature of the process.

Prognosis.—The disease is rapidly fatal, death occurring within periods ranging from two weeks to four months.

Treatment.—Complete rest, hypernutrition with liquid food, stimulants, and hydrotherapy are indicated. The persistent use of cold water, applying compresses to abdomen and trunk, may prove advantageous. The medicines are those recommended for the treatment of chronic tuberculosis.

FIBROID TUBERCULOSIS OF THE LUNGS.

Symptoms.—The onset is very gradual and marked by few symptoms, and the lesion may undergo permanent arrest without its existence ever having been suspected. Its presence may be indicated, however, by

1. *Hemoptysis*, which may be profuse and recurrent, without evidence of advance of the lesion.

2. *Cough*, which is rarely severe and is accompanied by little or no expectoration.

3. *Temperature.*—There is a more or less complete absence of evening pyrexia, with a tendency to subnormal temperature in the morning.

Physical Signs.—1. *Retraction* of the supra- and infra-clavicular regions of one side, with impaired expansion.

2. *Impaired resonance*, unless the latter be masked by emphysema.

3. *Breath sounds weak*, *expiration prolonged*, and *fine crackling rales*.

Diagnosis.—By physical signs. The latter may, however, be masked by emphysema, and in that case doubt can be removed only by discovery of the tubercle bacilli in the sputum.

Prognosis.—Fibroid tuberculosis tends to a prolonged course, if not complete arrest.

Treatment.—That of the ordinary chronic form.

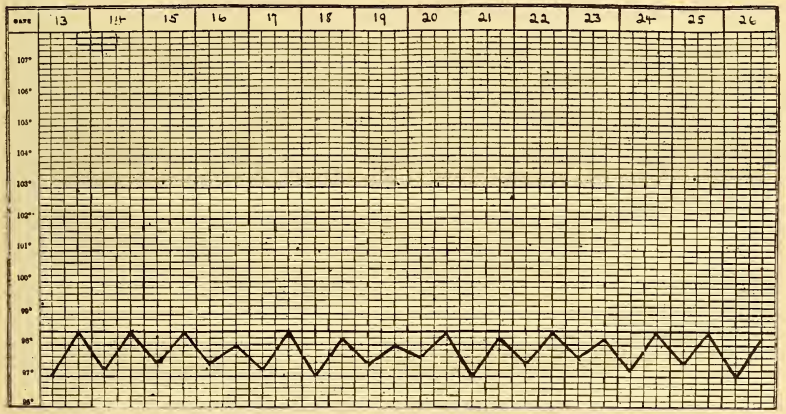

FIG. 11.—Chart showiug the Subnormal Temperature of Fibroid Tuberculosis.

CHRONIC PULMONARY TUBERCULOSIS.

(Fibro-Caseous or Ulcerative Tuberculosis.)

Symptoms.—1. The *mode of onset* varies greatly; frequently it is

(*a*) *Insidious*, the patient simply noting a gradual loss of strength with slight cough, little expectoration, anemia, and emaciation. Loss of appetite, dyspepsia, and vomiting after eating are common; and night sweats and dyspnea may be noticed.

(*b*) *Hemoptysis* may be the first indication of disease. It may be followed immediately by the usual symptoms of the disease, or the latter may be delayed for some time.

(*c*) *Pleurisy*, either dry or with effusion, often precedes the development of the lung lesion.

(*d*) *Bronchitis*, recurring in acute attacks for several years, particularly each winter, may finally be followed by evidences of tuberculosis.

(*e*) *Laryngitis*, though usually secondary, may occur before the lung infection. The patient complains of irritation in the larynx, cough, hoarseness and mucoid expectoration.

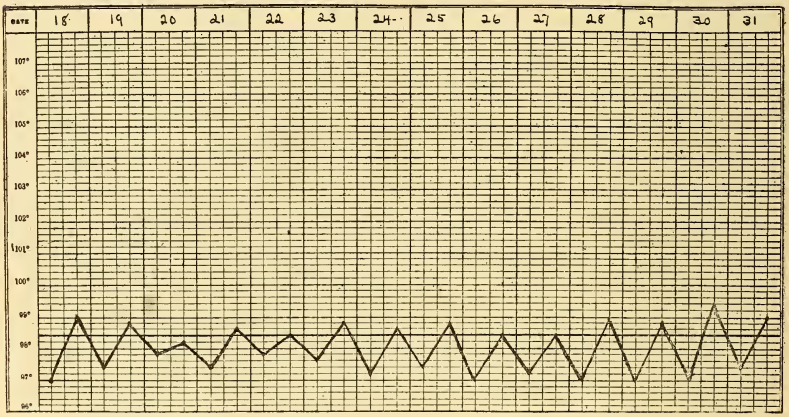

FIG. 12.—Chart showing the Temperature in a Quiescent Case of Chronic (fibro-caseous) Tuberculosis.

2. *Cough* begins early in the course of the disease. At first it is dry and hacking; later it is accompanied by expectoration and may occasion vomiting and dyspnea.

3. *Expectoration.* In the earlier stages the sputum consists of mucus, sometimes rendered watery by admixture with saliva. When softening occurs the sputum becomes purulent, yellow or green in color, and often blood-stained.

4. *Chest pain* is common; it may be due to pleurisy, intercostal neuralgia or myalgia.

5. *Pyrexia* may vary in its type, according to the activity

of the disease, and fever may be absent during long periods in which the disease is latent. During advance of the purely tubercular lesion the evening rise varies from 99° to 100.5°; but with secondary infection and softening the evening temperature is apt to rise much higher, with or without marked remissions to indicate its septic character. The pulse is rapid and weak throughout the disease.

6. *Emaciation* and *anemia* are generally coincident with the progress of the lesion; a gain in weight is therefore suggestive of arrest of the process. The anemia is of symptomatic type and leucocytosis is absent as a rule, although it may be observed when suppuration is in progress.

7. *Gastro-intestinal symptoms* are frequently prominent. The tongue may be coated, appetite lost, and thirst and other symptoms of gastric catarrh present. Vomiting may be the result of gastritis, but in many cases it simply follows severe coughing paroxysms. Diarrhea may result from dietetic errors, but when it occurs late in the disease it generally indicates intestinal ulceration.

Physical Signs.—During the stage of infiltration, before any extensive consolidation has developed, the more important physical signs, generally, but not always, heard at one apex, are:

1. *Feebleness* of the respiratory sounds, with or without cog-wheel breathing.

2. *Prolonged and high-pitched expiration* (broncho-vesicular breathing) often associated with *localized dry rales*.

When considerable solidification has occurred, the signs include, in addition to more marked broncho-vesicular breathing,

3. Depressed surface, *impaired movement* during respiration and evident loss of flesh, generally.

4. *Percussion dulness* over the diseased area.

5. *Increased* tactile and vocal *fremitus;* increased intensity of the whispered voice is especially significant.

L. of C.

When softening begins in the consolidated area—

6. The *rales become* larger, more numerous, and *moist.* (Moist rales may also be due to the presence of fluid blood; this must be excluded.)

When the process of softening and discharge has produced a cavity of large size, it may be recognized by—

7. *Cracked-pot resonance* on percussion.

8. *Amphoric or cavernous breathing.*

9. *Coarse, gurgling rales.*

Confirmatory evidence of the existence of a cavity may be found in other signs, such as a tympanitic percussion note with greatly increased vocal resonance (pectoriloquy); Wintrich's change of percussion note, the latter becoming higher when the mouth is open; and Gerhardt's change of sound, the tympanitic note becoming deeper when the patient sits up.

It must be remembered that all stages of the disease and consequently all the above physical signs may be present in one lung at the same time.

Prognosis.—While a majority of these patients finally succumb to the pulmonary disease, in all but the advanced stages arrest of the lesion and comparative health for years is possible. In any given case a prognosis must be based upon consideration of the following factors:

1. The *constitution* and family history of the patient.

2. The *stage of the disease.* Arrest is more common in the stage of infiltration.

3. The *character of the general symptoms* and their rate of progress. A poor appetite, digestive disturbances, and evident nutritional failure render the outlook dark. Continued pyrexia, or the inverted type, are unfavorable, indicating the onset of miliary tuberculosis.

4. The presence of *complications*, such as laryngeal tuberculosis, diarrhea, albuminuria, or peritonitis, is of unfavorable import.

Diagnosis.—In advanced cases the diagnosis is rarely difficult,

but in the earlier stages it is often impossible to decide as to the nature of the case by symptoms and signs alone. At any period of the disease doubt is ended by the discovery of the tubercle bacillus in the sputum. For this repeated examinations may be necessary, and it is essential that the expectoration so examined shall have come from deep in the chest; for this purpose that first raised in the morning is best. Select from a thin layer of sputum one of the small caseous particles, transfer it by means of a platinum loop to a cover-slip, press down upon it a second cover-glass, and separate the two by sliding them apart. Allow the cover-glasses to dry in the air, and fix the spread by holding each cover-slip, face upward, between the fingers and passing it through an alcohol flame. Then holding a cover-glass, still face upwards, with a pair of forceps, cover the surface with the Ziehl-Nielsen solution of carbol-fuchsin (fuchsin, 1 gm.; carbolic acid crystals, 5 gms.; alcohol, 10 c.c.; distilled water, 100 c.c.), and pass it slowly over the flame until steam arises. Do this seven times, without allowing the fluid to actually boil. Then rinse it in clear water, and counter-stain in Gabbet's solution of methylene blue (methylene blue, 1 gm.; sulphuric acid, 25% solution, 100 c.c.) for a minute or two. Examine the mounted slip under a $\frac{1}{12}$-inch oil immersion objective, and the tubercle bacilli will appear as small red rods, pus corpuscles and other organisms being stained blue.

Treatment.—This must include:

1. *Prophylaxis.*—Prevent infection by destroying the sputum before it becomes dry, and disinfect all other discharges. Caution the patient against swallowing the sputum, lest other organs become infected. Do not allow a tuberculous mother to nurse her children. Let the patient occupy a separate room, if possible, and see that the latter is well aired and occasionally disinfected.

2. *General Hygiene.*—The patient must be clad from head to foot in warm but light clothing, wool being preferable to silk. He should have a tepid or cold sponge bath on arising in the morning, followed by forcible dry friction. Douching and

massage of the chest wall are often advisable. The patient should rest for half an hour or more after each meal, for a longer period at mid-day if possible, and in case there is persistent fever (above 100°) rest in bed must be absolute. The amount of exercise must be graduated according to the patient's strength and the stage of the disease. Special abdominal and respiratory exercises are advisable in the early stages.

3. *Climatic Treatment.*—The ideal climate for the consumptive must have an atmosphere free from gases, dust, and micro-organisms; it must be dry, and a high altitude is desirable because of its stimulating effect on respiration. On the other hand, high altitudes do not agree with weak, debilitated patients, especially neurotics, nor with those subject to frequent or severe hemorrhages. On this account, when the patient is sent away from home great care must be exercised in selecting his resort. As a rule, a suitable place can be found on the great plains bordering the Rocky Mountains, in Colorado, New Mexico or Arizona. It is essential that good food be obtainable. Climatic treatment can be carried on at home in many cases, the patient spending almost the entire period of daylight, summer and winter, at rest in the open air. A reclining chair, with suitable protection from the wind, and wraps suited to the temperature, render this possible. At night he sleeps in a room with open windows, coverings protecting him from cold and screens from draught. This method, combined with forced feeding, is the foundation of the sanatorium treatment; and can also be used with fair success in the patient's home.

4. *Dietetic Treatment.*—The patient should eat three full meals per day, drinking little and avoiding too much food at one time. The diet must be nitrogenous, consisting of meat, milk, oysters, fish and eggs. In addition he should take, at an interval of half an hour or so after each meal, a luncheon consisting of raw eggs, broken into a cup, seasoned with salt and pepper, and swallowed without stirring. In this way from 6 to 12 or more eggs can be taken daily. Pure water may be imbibed freely between meals.

5. *General Medicinal Treatment.*—Of the many remedies, those have proven most useful whose influence is upon nutrition, as, for example, *creosote*, *guaiacol*, the *hypophosphites*, and *strychnine*. The *iodine* compounds are extremely valuable: particularly *arsenic iodide* in strumous individuals with weak hearts and a tendency to diarrhea, and *antimony iodide* and *stannum iodide* in cases with free muco-purulent expectoration, the result of suppuration and excavation. Symptomatic indications may be found for many other remedies.

6. *Symptomatic Treatment.* — *Fever* requires no special treatment unless the temperature rise above 100°; in the latter case, the patient should be put to bed and given such remedies as *baptisia*, *ferrum phos.*, *quinine ars.*, or *silica*. Sponging with cold water, or even the wet pack, may prove advantageous.

Sweating requires the administration of such medicines as *agaricin*, *camphoric acid*, *ferrum ars.*, *phosphoric acid*, *picrotoxin*, *pilocarpine*, or *silica*. The patient may be benefited by sponging with a saturated solution of alum or some other astringent.

Cough, when essential to the removal of secretion from the bronchial tubes, must not be interfered with. In many cases, however, the cough is simply the result of irritation at some point in the respiratory tract, and its cause, *e. g.*, naso-pharyngeal catarrh, should receive appropriate local treatment. Inquiry into the habits of the patient may reveal the fact that his evenings are spent in an ill-ventilated room, or that he goes from a warm room to a cold bed, and a troublesome night cough results. Discovery and removal of such causes control the symptom. Rarely is it advisable to resort to an opiate; in that case, give *codeine*, gr. $\frac{1}{4}$, or *heroin*, gr. $\frac{1}{24}$ or $\frac{1}{12}$.

When expectoration is at all free, antiseptic inhalations are valuable. Of a mixture of equal parts of beechwood creosote, chloroform and alcohol, place 20 drops on the sponge of a Goodno or Yeo respirator, and inhale for 20 minutes; renew the solution at these intervals until an hour has been con-

sumed, and repeat the inhalations, one hour at a time, three times a day. Eucalyptus, guaiacol, or terebene can be substituted for the creosote.

Hemoptysis, if profuse, requires that the patient be placed at rest in a cool room in a semi-recumbent position. He may be given bits of ice to suck, and internally such remedies as *aconite*, *geranium*, *hamamelis*, *hydrastis*, and *hydrastinine hydrochlorate*. Dangerous hemorrhage, uncontrolled by these measures, may require the use of *atropin* or *morphine* hypodermatically.

Chest pain is usually controlled by quiet and the use of such remedies as *aconite*, *actea*, *bryonia*, *scillitin*, or *kali carb.* Bathing, massage, friction and counter-irritation may prove useful.

Gastro-intestinal symptoms are of unfavorable import. A liquid diet, consisting largely of milk or koumiss, should be directed until the symptoms abate, and the return to solid food must be gradual. *Arsenic*, *cinchona*, *copper arsenite*, *ferrum phos.*, and *ferrum ars.* are among the important medicines.

SYPHILIS.

(Pox; Lues Venerea.)

Etiology.—The specific organism has not yet been discovered. Infection may be:

1. Hereditary.

(*a*) From the father, if the latter's infection has occurred within three or four years.

(*b*) From the mother, if the latter be in the active stages of the disease; if the disease has been acquired some months previous to the conception, the child usually escapes. Moreover, the mother may be infected through the placental circulation, but a syphilitic child cannot infect its mother after birth (Colles' law).

2. Acquired.

(*a*) By inoculation during sexual intercourse.

(*b*) By "extra-genital" inoculation, as by infected instruments, cups, pipes, kissing, etc.

ACQUIRED SYPHILIS.

Stages of the Disease.—Three periods are recognized, *viz.:*

1. A *primary stage*, extending from the time of infection to the outbreak of constitutional symptoms (8–10 weeks), which is divisable into—

(*a*) An incubation period of from fourteen to twenty-days, which ends with the appearance of the primary sore.

(*b*) A seond incubation period extending from the time of appearance of the chancre to the development of constitutional symptoms.

2. A *secondary stage*, lasting from one to three years and marked by skin manifestations.

3. A *tertiary stage*, of indefinite duration, marked by inflammatory and destructive lesions (*gummata*) which may attack any tissue of the body.

Pathology.—At least five lesions are characteristic of syphilis, *viz.:*

1. The *initial lesion*, the chancre, which occurs at the point of inoculation and consists of a vesicle or papule, which becoming softened at its centre, forms an ulcer with a hard, gristly base.

2. The *lymphatic glands* adjacent to the primary lesion, and often those of the entire body, become enlarged. Suppuration occurs, however, only when there is a mixed infection.

3. *Mucous patches* (*moist papules*) occur in the secondary stage and consist of inflammatory infiltrations of mucous membranes or of the moist portions of the skin, *e. g.*, the gluteal folds. By hypertrophy they may form venereal warts, especially about the vulva and anus.

4. The *cutaneous syphilides* may be macular, papular, pustular, or squamous; their color is apt to be "coppery," they excite neither pain nor itching, as a rule, and their distribution is usually symmetrical.

5. The tertiary lesions consist of circumscribed inflammatory products (*gumma*) of varying size which tend to disintegrate, forming ulcers, or to become enveloped in granulations and connective tissue, the subsequent contraction of which forms *fibroid indurations*. These lesions may appear in connective tissue, bones, skin, muscles, the visceral organs, etc.

Symptoms. — PRIMARY STAGE. Following infection, from fourteen to twenty-one days pass without symptoms. Then at the site of inoculation a single *chancre* appears and persists for several weeks. When its edges are grasped between the fingers its peculiar induration ("*parchment induration*") may be easily recognized, although the latter is not invariably present. Enlargement of the lymphatic glands all over the body occurs in the course of the next two weeks, those nearest the site of inoculation being affected first.

SECONDARY STAGE. In the course of six or more weeks the patient develops *sore throat*, due to hyperemia of the fauces, shallow ulcers on the tonsils, or mucous patches; laryngeal ulceration may also occur. Associated with these lesions there is usually irregular *fever*, languor, headache, bone-pains, and anemia (*syphilitic cachexia*). Various *syphilides* now appear; the most common is macular, a diffuse roseola coming out over the trunk; papular, pustular, and tubercular lesions are apt to appear a little later. At the same time *falling out of the hair* may be noted, and iritis, choroiditis, or retinitis is not uncommon.

TERTIARY STAGE. A period of freedom from symptoms and apparent good health usually follows the secondary manifestations, and in many patients this is permanent. In less fortunate cases, however, at the end of a year or more *deep-seated syphilides*, gummatous nodules, and fibroid indurations develop. On the surface the syphilitic *rupia*, in which laminated crusts

cover a deep pustule, is common. *Gummata* may develop in the skin or subcutaneous tissues, the bones or cartilages, or the viscera, and undergo ulceration and sloughing; in this way the cartilage of the nose may be destroyed, with sinking in of the bridge. In the viscera, the gummata may undergo fibrosis, with puckering and deformity of the organ. The bone affections at this stage are marked by *nocturnal pains*.

Sequelæ.—Amyloid degenerations; sclerotic degenerations, such as locomotor ataxia; and arterio-sclerosis.

Diagnosis.—The primary sore is distinguished from the *chancroid* by the fact that the latter is usually pustular, is never indurated, is usually multiple, has no period of incubation, and is followed by no constitutional symptoms. Chancre of the lip has been mistaken for *cancer*, but the history, symptoms, and therapeutic test will soon combine to settle the question.

Various *skin eruptions* (psoriasis, lichen, papular eczema) may resemble the cutaneous syphilides; if the history and associated symptoms are insufficient for diagnosis, careful investigation will nevertheless disclose minor points of differentiation.

The syphilitic arthritis which may develop at the onset of the second stage, and the pains of the tertiary stage, must be distinguished from *rheumatism* and *gout;* this is accomplished by a consideration of the history, the associated symptoms, and the therapeutic test.

Prognosis.—Under proper treatment, good. As a rule recovery is complete in from two and a half to three years.

HEREDITARY SYPHILIS.

Symptoms.—All of the lesions incidental to acquired syphilis, except the chancre, can occur in the victim of hereditary syphilis. In addition there is a tendency on the part of the mother to repeated *abortions*, the aborted fetus in each case being shriveled, with skin exfoliated, and evidently some time dead; and if, perhaps after a series of abortions, a viable child

is borne, it is apt to be *wizened*, poorly developed, with bullæ upon hands and feet (pemphigus neonatorum), *fissured lips*, and *snuffles*. The child may be apparently healthy at birth, but before the fourth month symptoms of the malady usually appear. Characteristic *coppery patches*, *notched teeth*, and an appearance of premature old age may be noted. Later, should the patient outlive his childhood, he is apt to be stunted, deformed, and undeveloped, appearing much younger than he really is—a condition of so-called *infantilism*. In exceptional cases the symptoms of the disease are not dicovered until the age of puberty or even later.

Diagnosis.—The appearance in a child of less than five months of snuffles and skin eruptions usually suffices for a diagnosis.

Treatment.—*Prophylaxis*. The patient must be cautioned as to the danger of his infecting his associates, and every thing involving contact with others must be forbidden until treatment has been prolonged, and all symptoms have disappeared for a considerable time. If a woman develops syphilis while pregnant, active treatment must be begun at once and continued after birth of the child.

Primary stage. Excision of the chancre does not prevent constitutional infection and is therefore inadvisable. Dust the sore with aristol, bismuth subnitrate, or calomel; but unless diagnosis is positive, administer no internal medicines until unquestionable secondary manifestations appear.

Secondary stage. Administer one grain of *mercury biniodide* 2x or two grains of *mercury protoiodide* 2x three times a day, after meals; continue this, and if necessary increase the dose, until the symptoms yield. Then prescribe the "tonic" dose of half that amount until active symptoms again appear, when the dose must be increased immediately to its former size. Should the stomach rebel, resort to inunctions: use one-half drachm of mercurial ointment mixed with an equal quantity of vaseline, daily. Spend from 20 to 30 minutes in rubbing it into the skin, selecting a different part of the

body for each inunction,—the axillæ, the groins, the inside of the thighs, etc. The limit of toleration on the part of the patient is indicated by increased saliva, a blue line on the gums, colic, and diarrhea; the dose must be kept just below this action until the symptoms yield.

During the period of active symptoms the patient must live a rational life, free from excesses of all kinds, and as far as possible he should be out of doors. His diet should be ample but easily digestible, daily or bi-weekly baths are beneficial, and he should have plenty of recreation. Smoking and chewing must be prohibited because of the tendency to lesions in the mouth; and the teeth must be kept in perfect order.

Tertiary stage. The tertiary manifestations have a specific remedy in *potassium* (or *sodium*) *iodide;* a saturated solution of this drug should be given, well diluted with water or milk, half an hour after each meal. The initial dose may be five drops three times a day, but this should be increased rapidly and if necessary to enormous quantities, in order to control the disease. Exceptionally the coincidence of skin and visceral symptoms will require the use of mercury and an iodide together (mixed treatment); in these cases it is well to give mercury by inunction and potassium iodide by the mouth.

Hereditary syphilis is treated in the same way, the doses of medicine being, of course, adapted to the age of the patient.

LEPROSY.

(Lepra.)

Etiology.—The *exciting cause* is the lepra bacillus, a minute rod closely resembling the tubercle bacillus. The disease is transmitted by contact, possibly only by inoculation, and generally as an incident of sexual intercourse.

Pathology.—1. Nodular infiltrations (tubercles) appear upon the skin, projecting from its surface; and since they are poorly supplied with blood-vessels, they soon undergo caseation and

either absorption or fibrosis, or else pyogenic infection leads to ulcerative and destructive changes. Similar lesions involve the mucous membranes and internal organs.

2. Nodular infiltration of various nerve trunks leads to neuritis, at first attended by local hyperesthesia, but later leading to complete anesthesia together with trophic changes in the parts supplied by the affected nerve. The nerve trunk, infiltrated with the leprous growth, may be felt as a thickened cord beneath the skin.

Forms.—According to the predominance of one or the other of these lesions, two forms of leprosy are recognized, viz., the *tubercular* and the *anesthetic*.

Symptoms.—The disease develops only after a prolonged incubation (3–5 years). In the *tubercular form* there appears at first a patchy redness of the skin which is slightly elevated and hyperesthetic. In a little while the redness and later the pigment disappear from the area, leaving it white and anesthetic (*lepra alba*). Tubercular nodules develop in the skin, the smaller ones disappearing again but the larger breaking down into ulcers which finally cicatrize and occasion marked deformity. Similar ulcerations affect the mucous membranes of the nose and throat.

In the *anesthetic form* the onset is marked by the development of painful hyperesthetic patches in the skin. Small bullæ, representing trophic changes, appear on the arms and legs, and the muscles supplied by the affected nerves undergo atrophy. Red patches of vasomotor congestion appear and in turn become anesthetic, and finally only dry, scaly, whitish patches remain. The trophic changes may become extreme, the bullæ bursting and leaving perforating ulcers, and as the result of atrophy and necrosis combined, great deformities may be produced, and fingers and toes may even drop off (*lepra mutilans*).

Diagnosis.—Patchy erythema with hyperesthesia, followed by local anesthesia and the appearance of characteristic nodules, affords unmistakable evidence of leprosy. *Syringomyelia*,

the only disease notably similar, is dependent upon a central rather than a peripheral lesion; its symptoms are localized to an area enervated from one spinal cord segment rather than from a nerve-trunk, and the tactile sense is not lost as in leprosy.

Prognosis.—The disease is hopeless, but the course is prolonged, and the patient may live in comparative comfort for many years. Death finally occurs from exhaustion or some intercurrent disease.

Treatment.—Symptomatic.

THE PLAGUE.

(Bubonic Plague; Black Death.)

Etiology.—The *exciting cause* is a bacillus, a short rod with round ends which gains entrance to the body through the digestive or respiratory tracts or through breaks in the integument. The organism is said to be conveyed by rats, and insanitary conditions predispose to the contagion.

Symptoms.—The onset of the disease is attended with severe pains in the head, back, and limbs, and intense dizziness. Sometimes there is a slight chill, and the temperature rises rapidly to a high point. There is thirst, often nausea and vomiting, and early delirium. Petechiæ, and in malignant cases hemorrhages from the mucous membranes, may be noted. If the patient does not succumb at the outset, on the second or third day the characteristic buboes appear, usually in the inguinal region; these may reach the size of an egg, are very tender, and often rupture and discharge. Upon the appearance of the bubo, the fever subsides, with a profuse sweat to signalize the crisis. Carbuncles may also be present.

Diagnosis.—*Typhus fever* presents a characteristic eruption and lacks the buboes, petechiæ, hemorrhages, etc.

Prognosis.—The prognosis is very grave, a large majority of those affected dying. Death may occur within a few hours, or life may be prolonged for a week or more. In favorable cases

convalescence is protracted by the profuse suppuration which occurs.

Treatment.—Prophylaxis is most essential; the infected individuals must be isolated, disinfection must be thorough, and hygienic defects must be remedied. The treatment must be conducted on general principles, including fever diet, symptomatic medicines, and antiseptic cleansing of the abscesses.

DISEASES OF THE CIRCULATORY SYSTEM.

In diseases of the circulatory system no definite relation between symptoms and physical signs exists. For example, the objective evidences of disease of the heart and arteries may be unmistakable while subjective symptoms are entirely absent. On the other hand, general symptoms may be dominant while the physical signs are slight or absent, as in angina pectoris. Finally, there are cases in which physical signs and subjective phenomena are closely associated, as in uncompensated mitral disease.

The one important question in any disease affecting the circulatory apparatus is the condition of the heart muscle. No matter how apparent a valvular defect, there will be no disturbance in the circulation so long as the cardiac muscle can by hypertrophy compensate for the deficiency. Hence, in every case the physician must determine not only what part of the heart is diseased, but to what extent the heart muscle is able to keep up the general circulation. In short, it is to the indications of cardiac failure that attention is generally directed. The cardinal symptoms of this are those due to *pulmonary congestion*, evidenced by dyspnea, cough, hemoptysis, or pulmonary edema; and those due to back pressure in the inferior vena cava, leading to *hyperemia of the liver*, *stomach*, *intestines*, *spleen*, and *kidneys;* and to general *dropsy*. Back pressure in the superior cava may induce cyanosis of the face and lips, and so little blood may be sent to the brain that vertigo or syncope is induced. These signs and symptoms

indicate grave weakness of the heart muscle, whether a valvular lesion be detectable or not.

Interrogation of the Patient.—When attention is directed to the heart or blood vessels, it is important to ascertain if there be—

1. A *family history* of gout, rheumatism, heart disease, Bright's disease, or paralysis of cerebral origin (hemorrhage, thrombosis, embolism).

2. A *personal history* of rheumatic fever, chorea, scarlet fever, diphtheria, syphilis, and other infections. In children, inquire concerning sore throats and "growing pains."

3. *Subjective sensations.* Does he suffer with dyspnea. Can he sleep lying down, or must he sit up in bed? Does he have any precordial pain or distress? What is its exact situation and character? Does it radiate or not? If so, in what direction? Does he complain of palpitation? What is its relation to meals and to exertion? Does the heart give an occasional thump? Is his sleep good or bad? Does he dream? Is vertigo ever present, and when?

4. *Signs of venous stasis.* Do the feet ever swell? Has he a cough? Is his digestion good? Does his nose ever bleed?

PHYSICAL EXAMINATION OF THE PATIENT.

Having placed the patient, stripped to the waist, in a good light, the observer should stand before him and systematically note the points elicited by inspection, palpation, percussion, and auscultation, concluding with an investigation of the pulse characteristics.

Inspection and palpation are usually practiced together, the examining hand verifying the discoveries of the eye.

1. Inspect the *precordia*, noting—

Its *form:* is there any bulging over the precordia? Is there any chest deformity which can displace the heart?

Its *movements:* note with the eye the *apex beat*, its situation, force, and diffusion. Place the flat of the hand over the cardiac area, the fingers covering the apex region, and notice

whether there is the normal moderate impulse, or the strong, heaving impulse of hypertrophy, the peculiar sudden tap of mitral stenosis, or the weak, perhaps imperceptible, slap of dilatation. Thrills and vibrations, systolic or presystolic, may be discovered at the same time. Note the *position* of the apex beat. Normally it is in the fifth interspace well within the nipple line; but hypertrophy of the left ventricle displaces it downwards, dilatation forces it outwards and downwards, and enlargement of the right ventricle pushes it outwards. Displacements may also be due to causes outside of the heart itself, such as pericardial adhesions, pleural effusion, etc., and these possibilities must be remembered.

2. Note any *epigastric pulsation.* This may sometimes be due to hypertrophy of the right ventricle.

3. Inspect the *aortic region*, noticing

(*a*) *Pulsation in the pulmonary area*, often seen in anemia.

(*b*) *Pulsation in the aortic area*, which may be seen and felt in cases of aneurism.

4. Inspect the *neck*, noting

(*a*) *Fulness of the jugular veins.* If there is distension of one side, it may be due to pressure (aneurism or tumor) on the innominate; if of both sides, it signifies either pressure on the superior rena cava or on both innominates, or dilatation and engorgement of the right heart.

(*b*) *Pulsation in the jugular veins*, signifying tricuspid regurgitation following great dilatation of the right heart.

(*c*) *Excessive pulsation in the arteries*, including the characteristic carotid throb below the ramus of the jaw which accompanies aortic regurgitation.

5. Inspect the *general circulation*, evidence concerning which may be found in the patient's complexion, his peripheral arteries and veins, the presence or absence of dropsy, etc.

Percussion verifies and extends the information gained through

inspection and palpation. With its aid the examiner investigates:

1. The *area of superficial cardiac dulness*. In health this is a small triangle whose internal border is formed by the left edge of the sternum, the external border being defined by a line running outward from the sternum along the lower level of the fourth cartilage nearly as far as its junction with the rib, and then dipping down and curving slightly outwards to the point of the apex beat. The lower border is lost in the liver dulness.

2. The *area of deep cardiac dulness*, which corresponds approximately to the true dimensions of the heart, should be determined by firm percussion at the end of expiration. Percussion must be made at successive points along lines approaching the dull area at right angles, until a change of note is detected.

The area of dulness may be extended to the left, passing the nipple line (dilatation of the left ventricle), to the right (dilated right ventricle), or in both directions and upwards as the result of pericardial effusion. In every case, however, it must be decided that there is actual enlargement, not displacement, of the heart, and not withdrawal of the adjacent lung, whereby a greater cardiac area lies against the chest.

3. The *region of the aorta* lies above the third cartilage. Dulness in this situation is always abnormal (aneurism, tumor of mediastinum).

Auscultation completes the examination of the heart itself, confirming or interpreting the evidence obtained by inspection, palpation, and percussion. The ear may be applied directly to the chest, or the examiner may prefer to use a stethoscope. The points of greatest importance, each representing the area at which the sounds produced at a certain valve are heard with greatest intensity, are four in number, viz.:

1. The "*mitral area*," at the apex of the heart in the fifth left interspace near the nipple.

2. The "*pulmonic area*" in the second left interspace near the sternum.

3. The "*aortic area*" in the second right interspace near the sternum.

4. The "*tricuspid area*" at the bottom of the sternum near the ensiform cartilage.

At each of these the examiner listens in turn. In health he hears characteristic normal sounds at each point. In disease there may be alterations in the character of those sounds, or there may be in addition certain adventitious sounds due to diseased conditions about the valve which obstruct the flow of blood (*stenosis*), or allow it to leak backwards (*insufficiency*, *incompetency*, or *regurgitation*). In the study of such an adventitious sound, called a *murmur* (*bruit*, *soufflé*), five facts must be ascertained:

(1) Its *time of occurrence* (during systole, during diastole, etc.).

(2) Its *point of maximum intensity*.

(3) The *area over which it can be heard*, *i. e.*, its direction of transmission.

(4) The *effects of position*, *respiration*, and *exertion* upon it.

(5) Its *character*, whether blowing, rasping, musical, etc.

Murmurs are loud in proportion to the force of the blood current, and therefore loudness is often a favorable sign, indicating a strong heart muscle. With dilatation and failure of compensation the murmurs, even though due to extensive lesions, may completely disappear.

1. In the **mitral** area both heart sounds are audible in health, the first long and low pitched and the second short and sharp. It may be noted that the first sound is *weaker* than usual (myocardial degeneration or asthenia), or it may be *louder*, *i. e.*, *accentuated;* in the latter case it will be short and sharp if there is dilatation, prolonged and dull if there is hypertrophy. Often the first sound is *reduplicated*, owing to lack of synchronism between the ventricles. The duration of the

interval between the first and second sounds, indicating the length of cardiac systole, and that between the second and first, representing the cardiac diastole, should be noted. The systolic interval may be so shortened that the second sound follows the first almost without pause; this indicates incomplete contraction of the ventricle and is often a grave sign, indicating impending heart failure.

The murmurs heard at the apex may be systolic, pre-systolic, or diastolic. The systolic murmur is usually smooth and blowing, sometimes rumbling, and signifies *regurgitation* through the mitral orifice. The pre-systolic murmur, so-called because it precedes and runs up to the first sound, is usually vibratory in character and indicates obstruction at the mitral orifice (*mitral stenosis*). If a diastolic murmur is heard at the apex it is usually one transmitted from the base.

2. In the **pulmonic** area organic murmurs may be systolic (*stenosis*) or diastolic (*regurgitation*), but these affections are rare, generally congenital. A majority of the murmurs heard in the pulmonic area are functional or else are simply transmitted from their origin at the mitral or aortic orifices. Accentuation of the pulmonic second sound is usual in children; in adults it is generally pathological and points to obstruction to the pulmonic circulation (mitral disease, emphysema, etc.).

3. In the **aortic** area there may be a systolic murmur, indicating stenosis, or a diastolic murmur significant of regurgitation. In addition, there may be accentuation of the aortic second sound as the result of any cause (arteriosclerosis, interstitial nephritis) which increases the resistance in the arterial system and so adds to the work of the left ventricle.

4. In the **tricuspid** area systolic, pre-systolic, and diastolic murmurs may be heard. The two latter are usually transmitted from apex or base and cannot be relied upon for diagnostic purposes; the systolic murmur may indicate tricuspid regurgitation.

Functional (*hemic*) *murmurs*, not due to any organic lesion of valves or orifices, are common. They are usually systolic,

mostly pulmonic, and are soft in character and not transmitted; they are remarkably evanescent, are unassociated with enlargement of the heart, and the patient is often anemic.

Exocardial sounds, due to pericardial or pleural friction, are distinguished by the fact that they are peculiarly superficial and are not synchronous with the heart beat. The pericardial sound is often heard only at irregular intervals. The pleuritic sound ceases when the patient holds his breath.

The Pulse.—It is customary to feel for the pulse in the radial artery simply because the wrist is most accessible; the facial or the temporal artery affords the same information and may be utilized occasionally. It is well to feel both radials at the same time, laying the tips of three fingers upon each artery. Note:

1. The *pulse rate*. In healthy adults this varies between 60 and 80; in children it is 100 or more.

2. The *pulse rhythm* (regularity, irregularity).

3. The amount of force necessary to obliterate the pulse (*compressibility*). To estimate this, gradually increase the pressure of the finger nearest the patient's heart until the pulse wave ceases to be felt by the other fingers. Experience is necessary in order to draw the proper conclusion.

4. The extent to which the artery collapses between beats (*tension*). The pulse of low tension completely collapses between beats, but that of high tension is perceptible between beats as a distinct cord which can be rolled between the fingers.

5. The *state of the arterial walls*. Flatten the vessel and cause the patient's skin to slip up and down over it; in health the vessel wall can scarcely be detected, but if it is diseased the thickening or tortuosity can be plainly felt.

In order to secure a permanent record and to aid in the analysis of the pulse characteristics, the **sphygmograph** may be employed.

The most convenient instrument for clinical use is that of Dudgeon. Slips of paper procured from the instrument maker

or else carefully cut to fit the sphygmograph and prepared by smoking one surface in the fumes of camphor, should be placed in the holder which accompanies the instrument. Find on the wrist the exact spot where the radial pulse is most distinct and mark it with an aniline pencil. Let the patient take a comfortable position, with the elbow resting on some solid support, such as the edge of a desk, and with the hand outstretched, palm upward. Place the metal button which works the writing lever exactly on the mark over the radial artery, tighten moderately the strap which holds the instrument in place, and endeavor to begin with the minimum pressure that will give characteristic tracings. Place one of the prepared slips of paper between the rollers, start the clockwork, and either stop it again before the slip has quite run out or else catch the latter in the hand. It is well to take three or four tracings, varying the pressure each time by turning the regulating lever until the greatest amplitude of movement is secured.

After the tracings have been made, mark with a pin-point on the smoked surface the name, date, etc., and dip the slip into a solution of gum benzoin, one ounce to six ounces of alcohol, and allow it to dry. It can then be preserved indefinitely.

The normal pulse tracing exhibits a straight, nearly vertical upstroke; a moderately acute apex; a gradual descent; a small tidal wave; and a well-marked dicrotic wave. Variations from this normal standard will occur in correspondence with changes in the force, volume, tension, and rhythm of the pulse; but for their correct recording and interpretation extended experience with the sphygmograph is necessary. The chief value of the instrument lies in the permanent record of the pulse characteristics which it furnishes.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Etiology.—Inflammation of the pericardium may be due to:

1. The toxic products of infectious disease (rheumatism, pyemia, scarlet fever, typhoid fever, etc.).

2. The accumulation of excrementitious matters in the blood (Bright's disease).

3. The extension of inflammation from the adjacent structures (pleurisy, tuberculosis, etc.).

4. Traumatism (blows, wounds, fractured ribs).

Pathology.—1. *Dry pericarditis.* The pericardial membrane become hyperemic, its epithelium detached, and its surfaces spread with yellow flakes of inflammatory lymph. By the incessant contact and separation of these sticky surfaces the "shaggy" or "hairy" heart is produced.

2. *Pericarditis with effusion.* Should the inflammatory process continue, an exudate of clear, straw-colored fluid is poured out and distends the sac. In favorable cases this is ultimately re-absorbed, leaving behind it *adhesions* between the pericardial surfaces. Rarely the exudate becomes *purulent.*

Symptoms.—Pericarditis is usually secondary to other diseases, and in consequence its onset is insidious. The patient complains of *pain* or more often *discomfort* in the region of his heart, and *dyspnea.* There is moderate fever (below 103°), the pulse becomes frequent, and there may be *tenderness* on pressure over the heart. As the effusion accumulates and oppresses the heart, the latter becomes irregular or intermittent, its second sound may be reduplicated, and during inspiration the pulse may become feeble or even cease (*pulsus paradoxicus*).

Physical Signs.—1. *Dry stage.* Pericardial friction, a rough "to and fro" murmur, heard irregularly during the greater

part of each cardiac cycle and increased in intensity by the pressure of the stethoscope.

2. *Stage of effusion.* (*a*) Extension of the area of cardiac dulness to both sides and upwards, forming a pyramid with its base in the nipple line and its apex about the first rib.

(*b*) Disappearance of the friction sound, with progressive muffling of the heart sounds, the latter disappearing first at the apex and last at the aorta.

(*c*) Obliteration of the intercostal spaces in the precordia, while the apical impulse becomes diminished or lost.

3. *Adherent pericardium* can rarely be diagnosed during life unless there are in addition adhesions to the chest wall or the adjoining pleura. In such cases the heart may be enlarged, and its apex fixed so that it does not alter its position during respiration or on change of posture; there may be retraction of the chest wall during systole, and on palpation a diastolic shock may be felt.

Diagnosis.—The friction sound of *pleurisy* is synchronous with the respiratory movements. *Endocardial murmurs* are less superficial and are not increased by pressure of the stethoscope. In *dilatation of the heart* the sounds are weak rather than distant or muffled, and venous stasis is more pronounced. If it is suspected that the pericardial effusion has become *purulent*, diagnosis can be rendered positive by the use of the exploring needle.

Prognosis.—Death rarely occurs, and uncomplicated cases may recover within three weeks. Pericardial adhesions may permanently cripple the heart, however.

Treatment.—Place the patient at rest, and apply hot poultices or ice-bags to mitigate the pain if it is severe. During the dry stage, *spigelia* is usually indicated, though *aconite* or *cannabis* may be preferred. When effusion occurs, *arsenicum*, *bryonia*, *cantharis*, or *mercurius corr.* should be given, according to the indications. *Digitalis* may be required by a weak heart, and *potassium iodide* may be given to promote absorption of the exudate. If the effusion is so great as to embarrass

the heart's action, aspirate in the fifth interspace about one inch to the left of the sternum.

Hydropericardium (serous accumulation in the pericardium) may occur as the result of obstruction to the veins of the heart by valvular lesions or pulmonary disease, or simply as part of a general dropsy. Its physical signs are those of pericardial effusion, but it is not accompanied by fever.

Hemopericardium (blood in the pericardium) may result from rupture into the sac of an aneurism, rupture of the heart itself, or wounds of the heart. It is quickly fatal.

Pneumopericardium (air or gas in the pericardium) is a rare condition, usually the result of traumation (stab wounds) or perforation by a foreign body. Fluid and usually pus are also present, leading to peculiar splashing sounds. It is rapidly fatal.

DISEASES OF THE ENDOCARDIUM.

ENDOCARDITIS.

Etiology.—The disease is invariably due to *infection* of the endocardial membrane, the organisms being those of rheumatism, chorea, diphtheria, scarlet fever, tuberculosis, cancer, etc. Chronic valvulitis predisposes to recurrent acute infection.

Pathology.—As a rule the inflammation is confined to the valves (*valvulitis*), although the lining of the cavity may be involved (*mural endocarditis*). In adults the left side of the heart is usually the one affected; in embryonic life it is the right. The infected valve becomes covered with small grayish patches, "vegetations," consisting of fibrin and leucocytes deposited on a base of granulation tissue. The latter becomes organized and later undergoes cicatricial contraction, so that the valves become puckered and mis-shapen and sometimes glued together at their margins (*chronic valvulitis*). Less often the lodgment of pyogenic organisms leads to necrosis,

with the formation of ulcers. In any case vegetations are liable to be broken off and carried away in the circulation to lodge elsewhere as emboli.

Symptoms.—These are to a great extent masked by the pre-existing disease, of which endocarditis is but a part. The occurrence of *dyspnea*, *precordial oppression*, a quickened and perhaps irregular pulse, and a slight rise of temperature, may lead the attendant to suspect the onset of endocarditis.

Physical Signs.—A murmur, most often heard in the mitral but sometimes in the aortic area, is alone conclusive. Its appearance may, however, be preceded by some alteration in the first sound (prolongation or reduplication).

Forms.—1. *Acute simple endocarditis*, the benign form, which is attended with warty vegetations.

2. *Infective endocarditis* (ulcerative, diphtheritic, or mycotic), the malignant form in which septic infection of the valves occurs. In this the valvular phenomena are accompanied by chills, sweats, cutaneous emboli, and other septic symptoms, a typhoid state develops, and the patient usually dies.

3. *Chronic endocarditis*, comprehending a majority of the chronic valvular defects.

Diagnosis.—In the course of any infectious disease the heart should be examined frequently for evidences of endocarditis. The simple form is recognized by the history and physical signs, not by the symptoms. Infective endocarditis may be distinguished from the simple form by its septic temperature and symptoms, and in doubtful cases the blood may be examined for pyogenic organisms and for the evidences of leucocytosis. In *pericarditis* the rub is heard only in the precordia, is very superficial, and is intensified by the pressure of the stethoscope. *Functional murmurs* are usually basic and are not attended by circulatory disturbance.

Prognosis.—In the simple form death is rare, but generally the heart is damaged to an extent that can be estimated only after inflammation has subsided and compensatory changes

have developed. In the infective form the outlook is very grave, few cases recovering.

Treatment.—Place the patient at rest between blankets and limit the diet to nourishing liquids, avoiding an excess of fluid. Warm baths may be given, and pain may require the application of heat or cold to the precordia. In the early stage, *aconite*, *veratrum viride*, or *spigelia* may be given; and if these prove unsatisfactory, *iodine* or *spongia* should be considered. *Bryonia* is useful in many rheumatic cases, and *belladonna* in children. Alcoholic or other stimulants are required in moderation when circulatory failure threatens. As the attack subsides, such remedies as *aurum*, *graphites*, *iodine*, *kali iod.*, and *sulphur* may be given in order to lessen the extent of the damage to the valves. In the infective form the principal remedies are *arsenic*, *crotalus*, *lachesis*, and *secale*.

CHRONIC VALVULAR DISEASES OF THE HEART.

Etiology.—The majority of these lesions are the result of *endocarditis*, but occasionally the defect is due to atheromatous or calcareous *degeneration*, as when a chronic sclerotic process extends down the aorta and involves the aortic valves. *Syphilis* is often an important factor, while *muscular strain*, such as that induced by heavy lifting, is a rare cause. In many cases the valve-leaflets remain perfectly normal, but as a result of dilatation of the cavities the orifices become so enlarged that the cusps cannot meet across the opening, and so-called "*relative insufficiency*" ensues.

MITRAL INSUFFICIENCY.

Pathology.—In this, the most frequent of valvular lesions, the valve leaks and permits the blood to flow back into the left auricle during systole. In consequence the left auricle, unable to resist this backward flow, dilates. Then the blood, backing up in the pulmonary circulation and engorging the lungs,

throws increased work on the right ventricle and the latter hypertrophies. This may suffice to restore compensation for a time, but finally the right ventricle undergoes dilatation, the tricuspid valve becoming relatively insufficient, and the right auricle and the general venous system become engorged, with resulting symptoms of stasis (dropsy, and passive congestion of the stomach, liver, kidneys, and other abdominal viscera).

Physical Signs.—1. A blowing, rasping, or musical murmur, entirely or partially replacing the first sound of the heart, is heard at the apex and frequently also in the fifth interspace toward the axilla and at the posterior border of the scapula. The pulmonic second sound is sharply accentuated, the aortic less so.

2. In proportion to the accompanying enlargement of the heart the apex is pushed outward and downward, and the area of dulness is extended in the same direction.

3. The pulse becomes irregular both in rhythm and force.

MITRAL STENOSIS.

Pathology.—This lesion occurs most often in women (75%), especially in the tuberculous, and is frequently combined with mitral regurgitation. The valve-leaflets may coalesce, forming a funnel; or by their cicatricial contraction a mere slit—a "button-hole" orifice—may be left. In either case the blood is restrained from passing into the left ventricle, hypertrophy and dilatation of the left auricle ensue, and this is followed by the series of changes in the right heart and venous system which have already been described as the result of mitral insufficiency.

Physical Signs.—1. A prolonged, grinding murmur, sharply localized to the apex or just above it, is heard during diastole or just preceding systole, *i. e.*, *presystolic*, and terminates abruptly in a sharp first sound. This murmur is never functional. The pulmonic second sound is accentuated and often reduplicated.

2. A *pre-systolic thrill* is felt by the hand placed over the apex.

FIG. 13—Pulse Tracing from a Marked case of Mitral Stenosis. (Salinger-Kalteyer.)

3. The pulse is not altered when stenosis is moderate; when it is extreme, the pulse becomes small from lack of blood, and when compensation fails it becomes irregular.

AORTIC INSUFFICIENCY.

Pathology.—This lesion, the most frequent cause of sudden death from heart disease, occurs next in frequency to mitral insufficiency, and is often combined with aortic stenosis. Its cause may be endocarditis, but more often it is arterio-sclerosis, the disease creeping down the aorta and involving the valve-leaflets. As a result the valve does not close properly, the blood flowing back into the left ventricle during systole. The left ventricle, endeavoring to restore the balance, hypertrophies until the heart becomes enormous in size (*bovine heart*). Ultimately the tremendous pressure leads to incompetency of the mitral valve, which is followed by the results already described.

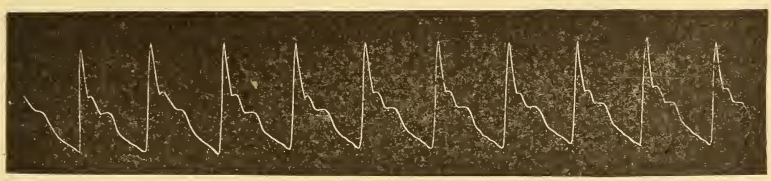

FIG. 14—Pulse Tracing From a Case of Aortic Insufficiency. (Eichhorst.)

Physical Signs.—1. A diastolic murmur, of variable characters is heard in the aortic area and transmitted down along the sternum.

2. Extension of the area of dulness and displacement of the apex downward and somewhat outward accompanies the hypertrophy.

3. A "water-hammer" (Corrigan's) pulse, in which the artery fills quickly but collapses immediately, is present. In addition there is violent arterial pulsation all over the body, most visible in the carotids.

AORTIC STENOSIS.

Pathology.—The valve cusps may be thickened and inelastic, so that they cannot fall back out of the way of the blood current, or they may be glued together, so that they obstruct the flow of blood. As a result the left ventricle hypertrophies, though not so enormously as in cases of aortic regurgitation,

FIG. 15—Pulse Tracing From a Case of Aortic Stenosis. (Eichhorst.)

and the sequential changes are the same. Uncomplicated aortic stenosis is the least common of valvular affections of the left heart, the lesion occurring more often in combination with aortic insufficiency.

Physical Signs.—1. A systolic murmur is heard in the aortic area and transmitted up into the arteries of the neck. This murmur is not pathognomonic of stenosis; it may be due to (1) anemia, (2) mere roughening or impaired flexibility of the valve, (3) acute or sub-acute aortitis, in which it is associated with substernal pain and heart failure (angina pectoris?), or (4) dilatation of the aorta just above the valves.

2. Enlargement of the left heart is demonstrable by percussion and by palpation of the apex.

3. The *pulsus tardus*, slow in reaching its maximum, small and frequent, but regular, accompanies aortic stenosis.

TRICUSPID INSUFFICIENCY.

Pathology.—This lesion is rarely primary, and if so it is usually congenital. As a rule it is a relative insufficiency, accompanying the dilatation of the right ventricle which is caused by back pressure through the lungs, and it leads to general venous stasis.

Physical Signs.—1. A systolic murmur, heard with greatest intensity about one-third of the distance between the left edge of the sternum and the nipple line, indicates tricuspid regurgitation. It is usually audible about the apex and may be mistaken for a mitral murmur.

2. The veins of the neck appear distended, often pulsating, and frequently they will fill from below when emptied by pressure.

3. The liver is congested and much enlarged, and may pulsate.

TRICUSPID STENOSIS.

Pathology and Physical Signs.—This rare lesion occurs in association with mitral stenosis, and is accompanied by a pre-systolic murmur heard in the tricuspid area and with difficulty distinguished from that of mitral stenosis. If the lesion causes considerable obstruction, symptoms of venous back-pressure ensue; and the latter, occurring early in a case of mitral stenosis, justify a suspicion of complicating tricuspid stenosis.

PULMONIC VALVE LESIONS.

Pulmonic Insufficiency occasions a diastolic murmur heard in the pulmonic area and transmitted downwards. It causes no special symptoms.

Pulmonic Stenosis is nearly always congenital and gives rise to a loud systolic murmur which is conducted along the branches

of the pulmonary artery over the entire chest. It is often associated with other congenital defects, notably a patent foramen ovale; in the latter condition cyanosis is usually present.

Diagnosis.—In the clinical study of valvular disease the following points must be considered:

1. The valve affected, and the relative danger attendant upon that particular lesion. For example, the liability to sudden death from aortic regurgitation is well known; of the other lesions, stenosis is usually more serious than a corresponding degree of regurgitation.

2. The origin of the lesion, whether in rheumatic or other endocarditis, or in degenerative changes, etc. The lesions due to endocarditis, occurring in the comparatively young, may be compensated for by hypertrophy; with degenerative lesions this is much less likely to occur.

3. The actual condition of valve and orifice, *i. e.*, the extent of obstruction or amount of regurgitation. This may be indicated by its effect on the heart, *i. e.*, by the degree of dilatation or hypertrophy induced.

4. The nutritional soundness and functional vigor of the heart muscle and of the body in general, in order to judge as to the extent and time during which compensation can be depended upon.

Prognosis.—The foregoing details, a knowledge of which is necessary to the proper diagnostic appreciation of a case, may be utilized in framing a prognosis, which implies consideration of—

1. The lesion, including
   - *a.* Its site and variety, with the relative danger attaching thereto.
   - *b.* Its extent.
   - *c.* Its character, whether progressive or stationary.
2. The patient, including
   - *a.* His nutrition, cardiac and general.
   - *b.* His age.

c. His family history, especially if there be hereditary tendency to heart disease.

d. His habits and surroundings.

e. The presence or absence of other diseases.

Treatment of Chronic Valvular Disease.—While compensation remains complete the physician should content himself with prescribing such hygienic regulations as the individual requires. If nutrition is good, allow as much gentle exercise in the open air as possible. The diet should be liberal, but stimulants must be taken only in moderation. A warm climate at a moderate elevation affords a desirable resort for these patients. They should be protected from exposure, and constipation must be avoided.

When symptoms of failing compensation are discovered, the patient must be put at rest, and a remedy prescribed in accordance with symptomatic indications. If these measures prove insufficient, then tincture of *digitalis* (gtt. ij — x t. i. d.) or one of its substitutes, such as *cactus*, *caffein*, *convallaria*, *spartein*, or *strophanthus*, must be prescribed. Should dropsy be present and persist in spite of these remedies, give *calomel* in doses of one-tenth grain every two hours for several days, or the infusion of *apocynum* or of *digitalis*, in doses of twenty drops or more every three or four hours. Sudden heart failure should be treated by rest, the application of heat to the precordia, and the hypodermatic use of *glonoin* 2x, gtt. i. — ij, or *strychnine*, gr. $\frac{1}{60}$—$\frac{1}{30}$.

After urgent symptoms have been relieved, it is well to resort to such remedies as *arsenic*, *arsenic iodide*, or *strychnine* to sustain the heart.

DISEASES OF THE MYOCARDIUM.

HYPERTROPHY OF THE HEART.

Etiology.—An increase in thickness of the heart muscle is invariably the result of increased activity of that organ, which may be induced by:

1. Obstruction within the heart (valvular diseases).

2. Obstruction outside the heart (aneurism, arterio-sclerosis, chronic nephritis, pericardial adhesions, etc.).

3. Long-continued functional activity induced by laborious exercise, athletics, prolonged tachycardia of nervous origin, etc.

Pathology.—When the cause lies at the aortic valve or outside the heart, the left ventricle is the portion hypertrophied; and in consequence this is the more frequent lesion. The right ventricle becomes involved when the obstruction lies at the mitral valve, in the pulmonary circuit, or in the right heart itself. The weight of the organ, normally about 12 ounces in men and 10½ in women, may become greatly increased as the result of added thickness of the walls. This increase in bulk is due to the development of new muscle fibres (numerical hypertrophy). Hypertrophy may occur without enlargement of the cavities (*simple hypertrophy*) or hypertrophy and dilatation may co-exist (*eccentric hypertrophy*).

Symptoms. — Hypertrophy is a compensatory process and occasions few symptoms. Occasionally a patient complains of precordial discomfort or actual pain, palpitation, and pulsation in the head or neck.

Physical Signs.—*Hypertrophy of the left ventricle* is indicated by an apex beat situated below the normal point and heaving in character, together with extension of the percussion dulness downward and to the left. The aortic second sound is louder than normal, but the first sound at the apex is duller. If the latter becomes prolonged, muffled, and "murmurish," careful auscultation is apt to show that it is reduplicated, thus revealing a tendency to dilatation.

Hypertrophy of the right ventricle is evidenced by displacement of the apex outward rather than downward, with extension of percussion dulness to the right of the sternum, and sharp accentuation of the pulmonic second sound.

Diagnosis.—By the physical signs.

Prognosis.—The only question is whether the hypertrophy has established adequate and durable compensation. It is adequate when no circulatory symptoms or distress on exertion remain. Impending failure of compensation may be indicated by a prolonged interval between the first and second sounds or by reduplication of the first sound.

Treatment.—Since hypertrophy is a conservative process, no direct treatment is required. The causative lesion should be controlled as far as possible.

DILATATION OF THE HEART.

Etiology.—Enlargement of the cavities of the heart may be due to—

1. Sudden over-distention, induced by excessive exertion or valvular deficiency (*simple dilatation*). This is usually followed by hypertrophy of the walls (*dilatation with hypertrophy*).

2. Thinning of the walls, owing to faulty nutrition. This represents the final stage, hypertrophy and the resulting compensation having failed.

Pathology.—The muscular tissue undergoes inflammatory or degenerative changes, as a result of which the wall becomes thinned and inelastic, and in consequence it is unable to cope with an increase of blood pressure, and dilatation of the cavities results. When nutrition can be maintained or restored, a compensatory thickening of the walls may follow; but when nutritional failure has ensued upon a previous compensatory hypertrophy the muscle rarely becomes competent again. As a rule, more than one of the cavities is enlarged, and stretching of the valvular orifices often gives rise to relative insufficiency. The left auricle and right ventricle are the cavities most frequently involved.

Symptoms.—The dilatation may be *acute*, the result of heart strain from over-exertion, or occurring in the course of an acute infectious fever. The symptoms are apt to consist of sudden pain about the heart, a rapid, feeble pulse, faintness,

and dyspnea. This attack may be followed immediately by death; if not, the symptoms may pass off while the patient is at rest, but until compensation is established they tend to reappear on the slightest exertion.

In the more *chronic* condition resulting from a gradual loss of compensation, the symptoms are usually those of general venous stasis (dropsy), together with feeble pulse, dyspnea, palpitation, and sometimes anginal attacks.

Physical Signs.—The apex is displaced and the area of percussion-dulness extended as in the case of hypertrophy, but the impulse is diffused and weak. The apical first sound is short and the aortic second sound is enfeebled. The interval between the first and second sounds may be lengthened, or if failure is imminent it may be shortened, owing to incomplete contraction of the ventricle. Pre-existing murmurs may disappear, and on the other hand a mitral systolic murmur, due to relative insufficiency, may be heard.

Prognosis.—Recovery from acute dilatation is possible; the chronic form is ultimately fatal.

Treatment.—Rest, a light nutritious diet, and the administration of heart tonics as directed for the general treatment of valvular disease, of which this is but a final stage.

ACUTE MYOCARDITIS.

Etiology.—Acute inflammation of the heart muscle is usually associated with peri- or endocarditis, and is due to the same causes, *e. g.*, infectious diseases.

Pathology.—The muscle fibres undergo changes varying from cloudy swelling to granular degeneration, while the connective tissue becomes proliferated. In connection with pyemia the process may be suppurative, the heart substance becoming infiltrated with pus. The consequent weakening of the myocardium leads to acute dilatation (*q. v.*).

Symptoms and Physical Signs.—These are indicative of progressive dilatation and asthenia, the latter being evidenced by

feebleness or loss of the apex beat, and a rapid, easily compressible pulse.

Prognosis.—This varies with the extent of the process. The milder cases recover; the more severe may recover after prolonged convalescence, but sudden death from slight exertion is not unusual. Suppurative cases rarely recover.

Treatment.—That of the causal disease.

CHRONIC MYOCARDITIS.

(Fibrous Myocarditis; Fibroid Degeneration.)

Etiology and Pathology.—As the result of disease of the coronary arteries the blood supply to the heart becomes diminished, and ultimately impaired nutrition leads to disappearance of the muscle fibres, which are replaced by connective tissue. The process may be diffuse or circumscribed.

Symptoms and Physical Signs.—These are identical with those of dilatation of the heart. A slow pulse and anginal attacks are common.

Diagnosis.—This is often difficult. The absence of a history of valvular disease, the existence of arterio-sclerosis elsewhere, and the occurrence of bradycardia and anginal attacks suggest fibroid degeneration.

Prognosis.—Grave.

Treatment.—Rest, nourishment, and the use of such drugs as *chloride of gold*, *iodide of arsenic*, etc. Syphilitic cases should receive *mercurius biniod.* or *kali iod.* *Digitalis* and other heart-whips are of little value.

DEGENERATIONS OF THE HEART MUSCLE.

Parenchymatous or albumenoid degeneration (cloudy swelling) occurs in infectious fevers and as a first stage of fatty degeneration.

Fatty degeneration (fatty metamorphosis, yellow atrophy) consists of a metamorphosis of the muscle-protoplasm into

fat; it may be circumscribed, occurring in small foci, or general, as in dilatation of the heart succeeding hypertrophy. It is not recognizable during life. Death is liable to come suddenly. When the disease is suspected, the general treatment should be that of dilatation, together with such remedies as *arsenic*, *aurum*, *iodine*, *kalmia*, and *phosphorus*.

Fatty infiltration consists of a deposit of fat between the muscle fibres. It occurs as a part of general obesity, to which treatment should be directed.

Atrophy of the heart consists of a wasting of the muscle substance and a reduction in the size of the cavities. It develops in the course of wasting diseases, such as tuberculosis and cancer. *Brown atrophy* consists of a similar wasting in which the heart muscle becomes pigmented; its causes are the same, and in addition it occurs in connection with senile marasmus and chronic valvular disease.

Amyloid degeneration may take place in the heart as it does in the other viscera.

NEUROSES OF THE HEART,

PALPITATION.

Etiology.—The subjective sensation of forcible or rapid beating of the heart may be due to:

1. Emotional causes, such as joy, sorrow, or fright.

2. Neurasthenic conditions, the result of overwork, excesses, etc.

3. Toxic causes, including the over-use of tea, coffee, tobacco, and alcohol.

4. Reflex conditions in the stomach and other viscera.

Symptoms.—The patient complains of violent beating of the heart, and on examination its action is often found to be rapid and forcible. In addition he may have sensations of fluttering and faintness, and there may be pain of anginal character.

Diagnosis.—The diagnosis, which is usually easy, rests upon

the exclusion of an organic cause for the symptoms. Search should also be made for the etiologic factors mentioned.

Treatment.—Rest, the application of cold or heat to the precordia, and therapeutic measures directed to the cause.

TACHYCARDIA.

Rapidity of the heart, the beat ranging from 100–200 or more per minute, may be symptomatic of many conditions, including various neuroses and lesions of the central nervous system, of the sympathetic system, and of the pneumogastric nerve. The tachycardia may be continuous or paroxysmal, and various subjective sensations may accompany it. In every case search should be made for the cause, whether it be a lesion of brain, cord, or thoracic viscera, or exophthalmic goitre, hysteria, or other neuroses.

BRADYCARDIA.

Unnatural slowness of the pulse, 60–40 or less, is often a symptom of serious degenerative change in the myocardium or aorta. Pulmonary, digestive, and renal disorders, toxic agents, and various lesions involving the medulla or cord, may also induce bradycardia. The beats should be counted while listening over the heart, for in some cases of ventricular failure every pulsation does not appear in the radial artery. The discovery of bradycardia should lead to a search for the cause, to which treatment must be directed.

ARHYTHMIA.

Cardiac arhythmia may be limited to simple *irregularity* of action, or there may be actual *intermission*, the heart dropping a beat at intervals. In the latter case it is important to determine whether the systole is omitted, the first sound being absent, or whether it is simply weak, not sending the pulsation to the wrist. Arhythmia may be the result of myocardial weakness, but more often it is reflex or neurotic. Extreme ir-

regularity is known as *delirium cordis*. *Embryocardia* (*fetal rhythm*) is distinguished by a beat in which the long pause is shortened and the character of the first sound modified, so that the two sounds are alike and follow each other like the ticking of a watch. It is an alarming symptom, indicating extreme cardiac dilatation or asthenia. "*Gallop rhythm*" is characterized by a reduplication of the first or second sound, so that the heart sounds resemble the footfalls of a cantering horse; it is sometimes of serious import, especially in chronic nephritis. The *bi-* or *trigeminal pulse* is so called because of separation of the beats into groups of two or three; it occurs in many heart affections, especially mitral disease.

ANGINA PECTORIS.

(Stenocardia; Neuralgia of the Heart.)

Angina pectoris is a term applied to certain paroxysms of heart pain or discomfort which occur as symptoms of various lesions more or less grave.

Etiology and Pathology.—Angina pectoris appears to be related to a number of degenerative lesions, especially obstruction of the coronary arteries from atheroma or thrombosis, myocardial degeneration, aortitis, and arterio-sclerosis. Its exact nature is unsettled; it has been ascribed to spasm of the heart muscle, to a general vascular spasm, and to a neuritis of the cardiac plexus.

Symptoms.—The paroxysm may be excited by exertion or emotion, but often occurs without apparent cause, even awakening the patient from sleep. The typical attack presents two striking symptoms, viz.,

1. *Pain* in or about the heart, frequently radiating into the left shoulder or arm. The character of this pain varies; in some cases it is mere discomfort, but in others it is an indescribable agony, as if the heart were being gripped by an iron claw and torn from the chest.

2. *A horrible sense of impending death.* The patient stands

motionless, not daring to move or breathe; his eyes are staring, his face bathed in a clammy sweat, and he feels as though every moment were his last.

The duration of the paroxysm varies from a few seconds to several minutes. At its conclusion there is usually an *eructation of gas* from the stomach, to the presence of which the patient frequently attributes the attack. Occasionally he vomits or passes large quantities of pale urine.

Forms.—An attempt has been made to distinguish—

(1) *Angina pectoris vasomotoria*, characterized by a spasmodic contraction of the cardio-vascular system; (2) *angina pectoris gravior*, in which cardio-vascular spasm is associated with a diseased heart; and (3) *primary cardiac angina*, in which the diseased organ is itself the seat of the disturbance. In addition, a *pseudo-angina* of neurotic origin is described. Clinically it is impossible to distinguish clearly between these varieties of heart pain.

Diagnosis.—The typical attack is easy of recognition; but if either the pain or the sense of impending dissolution is absent or not characteristic in degree, doubt must be felt as to the gravity of the seizure. As a rule, a high tension pulse, contracted during the paroxysm, indicates grave ("true") angina. The first attack is, moreover, almost invariably experienced during exertion. The attack of *pseudo-angina* in a neurotic patient is often prolonged and attended with restless excitement and hysterical symptoms. *Gastralgia* usually appears when the stomach is empty, the pain does not radiate to the shoulder or arm, the sense of impending death is absent, and heart disease is not necessarily associated with it.

Prognosis.—Recovery after typical attacks of angina is possible, but rare; death is common in the second or third attack. It is impossible to frame an accurate prognosis, but the coexistence of marked cardio-vascular degeneration renders the outlook dark.

Treatment.—The paroxysm requires the prompt use of *amyl nitrite*, a few drops being inhaled from a handkerchief, or one

or two drops of *glonoin* 2x may be placed on the tongue. Inhalations of chloroform or ether and the hypodermatic use of morphia are also recommended. Between paroxysms treatment should be directed to the conditions found in heart or arteries. Arterio-sclerosis suggests gout or syphilis, and the discovery of either of these causal factors affords a basis for treatment. The patient must avoid immoderate exercise and emotional excitement, must take small, frequent and nourishing meals, and must avoid constipation. Among the more important medicines are *arsenic*, *nux vomica*, and *phosphorus;* indications may also be found for *aconite*, *aurum*, *belladonna*, *cactus*, *kali iod.*, *spigelia*, and many other remedies.

DISEASES OF THE BLOOD VESSELS.

ARTERIO-SCLEROSIS.

(Endarteritis; Atheroma; Arterio-capillary Fibrosis.)

Etiology.—1. *Old age.* Senility is essentially a condition of arterio-capillary degeneration.

2. The presence in the blood of *toxic substances*, due to gout or its causes, such as over-eating and drinking and lead poisoning, to chronic nephritis, and to syphilis.

3. *Excessive work* combined with faulty nutrition and hygiene.

4. *Heredity.*

Pathology.—The sclerotic changes occur most frequently in the wall of the aorta; the coronary arteries and the other large arteries are also commonly involved. The disease may be circumscribed or diffuse.

1. *Circumscribed arterio-sclerosis.* In this form the intima, which normally is smooth, exhibits localized areas of thickening, yellowish-white in color, which may ultimately undergo softening and disintegration (athermatous abscess). The disease begins as a localized infiltration of the outer coats of the vessel, the connective tissue hyperplasia in the intima being

a compensatory process. These areas may become disintegrated before thickening of the intima has supervened, and local dilatation is the result.

2. *Diffuse arterio-sclerosis.* In this form, which is often associated with circumscribed athermatous patches in the aorta, the intima appears smooth, but microscopic examination reveals great thickening of it and the other coats with connective tissue, the muscle fibres in the media being degenerated or destroyed. Calcareous deposits may render the artery of bony hardness.

Sequelæ.—The weakened vessel wall is predisposed to dilatation (aneurism of aorta; miliary aneurisms of cerebral arteries). The thickened walls offer resistance to the blood current, thus increasing the pressure and inducing hypertrophy of the left ventricle. The reduced lumen of the vessels, by lessening the blood supply to various organs, occasions secondary sclerotic changes in the latter (interstitial nephritis, fibrous myocarditis, hepatic cirrhosis, pulmonary emphysema, etc.).

Symptoms.—Various superficial vessels, especially the temporal arteries, appear *dilated and tortuous;* and a similar condition may be demonstrated in the radials by palpation. Subjective symptoms, when they occur, are due to sequential changes in the *cerebral circulation* (vertigo, head-pains, intellectual failure, and hemiplegia from embolism, thrombosis, or hemorrhage), in the *heart* (symptoms and physical signs of hypertrophy or dilatation), in the *kidney* (evidences of nephritis), or in *peripheral circulation* (dry gangrene).

Diagnosis.—The association of high arterial tension, hypertrophy of the left ventricle, accentuation of the aortic second sound, and thickened arteries affords positive evidence of arterio-sclerosis.

Prognosis.—The condition is irremediable, but the patient may nevertheless live to an advanced age.

Treatment.—Treatment must be directed to the causes, gouty or otherwise. The diet should be simple, nourishing, and non-stimulating. Active exertion must be avoided, and the hours

of sleep prolonged. The skin should be kept active by frequent bathing, and constipation must be avoided. The principal medicines are *aurum*, *cuprum*, *plumbum*, and the *iodides*. *Glonoin* 2x, in drop doses, may be used to lessen the high arterial tension. Complicating conditions will suggest other remedies.

ANEURISM.

Etiology.—Dilatation of a portion of an arterial wall may be due to:

1. Arterio-sclerosis, and hence the causes of the latter, notably gout and syphilis.

2. Sudden intense strain. Most cases of aneurism originate during the period of greatest physical activity, between the thirtieth and fiftieth years of life; but it is probable that strain is effective only when arterial degeneration pre-exists.

3. Embolism of a vessel, if complete, may be followed by aneurismal dilatation at its proximal side.

4. Parasites may induce local arterial changes which lead to dilatation.

Pathology.—A fusiform or cylindrical aneurism is simply a dilatation of an artery at some point. A saccular aneurism is due to the bulging of a circumscribed portion of an arterial wall as the result of disease or injury; in the sac thus formed the intima is ruptured, the media atrophied, and the wall consists of the adventitia, fibrous tissue, and adherent surrounding strictures. It contains laminated clot, which may later become organized.

ANEURISM OF THE THORACIC AORTA.

Aneurism involves the thoracic aorta in 75% of all cases, the abdominal aorta and its branches being affected in but 25%. Nearly two-thirds of the thoracic cases occur in the ascending portion, and most of the others in the arch.

Symptoms.—1. *Pain* is usually present. It varies, according to the site of the aneurism, from vague distress to intense boring and gnawing sensations and even anginal attacks.

2. *Pressure symptoms.* These may be due to—

a. Pressure upon the great veins. Sudden compression of the vena cava may occasion edema of the head, neck and upper extremities, with dyspnea and cyanosis. Gradual compression is met by the establishment of collateral channels, which may appear as tortuous veins on the surface of the chest. The jugulars may become distended and are not emptied by a deep inspiration, nor do they pulsate, as when the distention is due to venous back-pressure. Obstruction of one innominate vein may occasion unilateral distention of the internal and anterior jugulars, which does not disappear on deep inspiration.

b. Pressure upon the root of the lung. Pressure on one bronchus permits the entrance of only a certain amount of air, and this may occasion changes in the breath sounds. There may be pressure on the pulmonary veins, causing great congestion of the lungs; on the cilio-spinal branches of the sympathetic, causing dilatation or contraction of the pupil on the corresponding side; or on the left recurrent laryngeal nerve, causing paralysis of the left vocal cord (hoarseness).

c. Pressure on the trachea (dyspnea), esophagus (dysphagia), or thoracic duct may occur.

Physical Signs.—1. *Pulsation, expansile in character*, in an abnormal situation (usually above the third rib) can sometimes be seen and otherwise may be felt.

2. A characteristic *vibratory thrill*, and a *diastolic shock* at the end of the pulsation, may be felt.

3. A *tracheal tug* may be detected. The examiner, standing behind the patient, supports the latter's head against his body, and places the tips of his fingers beneath the cricoid, putting the trachea gently on the stretch. A short tug, felt with each cardiac systole, is an important sign of aneurism or dilatation of the aorta.

4. *Dulness* on percussion in a suspected area may be the most important early sign of aneurism.

5. A *loud, low-pitched aortic second sound* may be heard.

6. A *murmur* may or may not be present.

7. The *pulse* of one side may be smaller than the other, or it may be delayed.

8. The auscultatory signs of pressure on the root of one lung may be discovered.

Diagnosis. — *Aneurism of the ascending arch* usually approaches or penetrates the chest wall about the second interspace just to the right of the sternum. It may occasion pain, dulness, pulsation, and thrill.

Aneurism of the transverse arch is usually recognized only by pressure symptoms (cough, hoarseness, tracheal tug, unequal pupils, etc.).

Aneurism of the descending aorta occasions severe, persistent pain in the back, and in advanced cases dulness and pulsation may be found in the region of the left scapula.

Aneurism of the abdominal aorta is associated with sharp pain in the back, often increased by eating and drinking, and various gastro-intestinal symptoms; the femoral pulse may be delayed, and a pulsating, expansile tumor may be discovered. This must be distinguished from the pulsating aorta of nervous women, which presents neither pressure symptoms nor definite tumor. An *abdominal tumor* may receive pulsation from the adjacent aorta, but it is non-expansile, and is accompanied by cachectic and other characteristic conditions.

Prognosis.—The course of aneurism may be prolonged over several years, but it ultimately proves fatal, either by rupture or gradual circulatory failure.

Treatment. — This is unsatisfactory. Various mechanical measures, such as galvano-puncture or the introduction into the sac of foreign substances, such as silver wire, have been used in an effort to induce obliteration of the sac by clotting. A more popular method is that which places the patient at rest,

upon a light, dry diet, and administers *potassium iodide*, gr. x–xx, three times a day for a period of weeks. Good results have in a few cases followed the hypodermatic injection at weekly intervals of a few c.c. of a solution of gelatin (1–5%) in a normal saline solution. These measures are calculated to increase the coagultability of the blood and thus favor clotting within the sac of the aneurism.

DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE UPPER RESPIRATORY TRACT.

Diseases of the upper air passages (nose, throat, and larynx) may be indicated by a variety of symptoms. Attention is frequently attracted to the nose by *discharge*, *pain*, and *occlusion*, and nasal disease is a common cause of frontal *headache*, especially when the frontal sinus is involved. *Neuralgia* may be due to disease of the accessory chambers, notably the antrum. *Epistaxis* is often the result of local conditions, but its constitutional causes (anemia, arteriosclerosis, etc.) must not be overlooked. *Mouth-breathing* results from occlusion of the nose by hypertrophic catarrh, polypi, septal deviations, adenoid vegetations, etc.; and certain reflex neuroses, notably *asthma* and *hay fever*, can sometimes be traced to disease of the nose.

In most of these conditions the pharynx is involved. The voice may become *nasal* as the result of paralysis (post-diphtheritic) or ulceration (usually syphilitic) of the soft palate, or it may be due to total occlusion of the nose by catarrh or polypi.

Disease of the larynx also occasions *changes in the voice*, which may become hoarse, indistinct, or even lost.

Dyspnea may result from narrowing of the glottis (diphtheria, laryngitis, edema, or paralysis); it is inspiratory in character, often accompanied by stridor, and must be distinguished from tracheal stenosis, in which the larynx does not move in inspiration and the voice is not hoarse. Laryngeal *pain* is common but without special diagnostic significance. A

laryngeal *cough* is usually loud and barking ("croupy"), and *expectoration* may accompany it. *Dysphagia* occurs when disease of the larynx has involved the epiglottis and posterior wall.

CLINICAL EXAMINATION OF THE UPPER AIR PASSAGES.

The patient should be seated facing the examiner, with a light placed a little to one side of his head and on a level with the eye of the examiner. The latter adjusts a head-mirror to his forehead and manipulates it until the hole in its centre is directly opposite his right eye and the light is directed into the patient's nares.

1. **The nose.** Lift the tip of the nose with the finger, and note if there be any eczematous or ulcerated condition of the skin or mucous membrane, any dried secretion or blood, and any ulcer or perforation of the septal cartilage. Then gently introduce a speculum and separate the blades. As the inferior turbinated comes into view, notice whether it is enlarged; and if it appears to be, touch it with a probe to determine whether it consists of swollen mucous membrane or bone. Depress the patient's chin slightly to bring the inferior meatus into view, and then have him tilt his head back a little so that the middle meatus and middle turbinated can be seen. The superior turbinated can rarely be seen, the superior meatus never. Inspect the septum, searching for spines, deviations, ulcers, or perforations.

Then select the smallest laryngeal mirror and hold it face downwards over the lamp until a cloud passes over its surface, and touch it to the cheek or back of the hand to ascertain that it is not too hot. Have the patient depress his chin slightly and open his mouth, and introduce a tongue-depressor, holding it with the left hand. Grasping the mirror, like a pen, in the fingers of the right hand, introduce it, face upwards, behind the soft palate; and by rotating it a little the posterior nares can be seen. With the posterior end of the septum in the centre, on either outer wall the middle turbinated appears

as a bluish-red swelling, with the superior meatus and superior turbinated above it and the middle meatus and inferior turbinated below. Notice the size of each of these, its color, and the presence of mucus or pus. Then turn the mirror to face a little more directly upwards and to one side and look for the projecting bright red cushion which limits the depression leading to the orifice of the eustachian tube. Notice whether there is any secretion at the mouth of the latter. Finally, turn the mirror upwards and look for adenoid or other tumors at the vault of the naso-pharynx.

2. **The Pharynx.**—With the aid of the tongue-depressor those portions of the pharynx, soft palate, and fauces not comprehended in the nasal examination should be inspected. Note the color; the yellow tinge of jaundice or the patchy redness of measles may be discovered. Notice the presence of ulcers or mucous patches. Note whether the tonsils are enlarged, and whether they exhibit any grayish patches; if they do, try to wipe them off and see whether a healthy or a raw surface (diphtheria) is left. Examine the pharynx. Generally a number of swellings like sago-grains can be seen; if they are much increased there is granular pharyngitis. Note if there is any ulceration, any excess of mucus, or if there is any bulging (retro-pharyngeal abscess).

3. **The Larynx.**—Have the patient put out his tongue and grasp it between the folds of a clean, dry napkin, and withdraw it sufficiently to expose the vault of the pharynx. Introduce the mirror slowly, without touching tongue or palate, until, as the soft palate rises during inspiration, the back of the mirror can be placed at the vault of the pharynx. Then raise or lower the handle of the mirror until the back of the epiglottis comes into view. Tell the patient to say "a" and, as a rule, the vocal cords can be seen. Notice the color of the cords and mucous membrane, the presence of any swelling or ulceration, and the position and mobility of the vocal cords.

It is well to caution the patient in advance to breathe regularly through his nose; this engages his attention and often prevents gagging. In many cases the application of cocain

(5%) to the pharynx is a necessary preliminary to examination; and at best the latter is often unsatisfactory the first time.

ACUTE RHINITIS.

(Coryza; Cold in the Head.)

Etiology.—Acute catarrhal inflammation of the nasal mucous membrane may be primary, due to exposure to cold and wet or to inhalation of irritants (dust, chemicals), or secondary, a symptom of measles, influenza, etc. Depressed nutrition predisposes to attacks.

Pathology.—As a result of vasomotor dilatation the mucous membrane becomes red, swollen, congested, and covered with secretion, and the passages are obstructed.

Symptoms.—The attack usually begins with sneezing, and dryness and *obstruction* of the nasal passages, the latter often compelling the patient to breathe through his mouth. In a few hours a watery, sometimes irritating, *secretion* appears, and later this becomes muco-purulent. Mild febrile symptoms accompany the disease. The inflammation may extend to the frontal sinus, causing frontal headache, facial pains, etc.; it may occasion conjunctivitis, pharyngitis, laryngitis, bronchitis; or by involving the eustachian tube it may cause temporary deafness.

Prognosis.—Recovery within a week or two.

Treatment.—For the early stage, administer *aconite*, *belladonna*, *camphor*, or *gelsemium*. When secretion appears, *cepa*, *kali bichromicum*, *pulsatilla*, or *sulphur* may be indicated. If there is much obstruction, *ammonium carb.*, *lycopodium*, *nux vomica*, or *sticta* will be preferable. Sprays of albolene or other oils, plain or mentholated, are beneficial.

CHRONIC RHINITIS.

(Chronic Nasal Catarrh.)

Etiology.—Chronic catarrh of the nasal passages, characterized by a persistent muco-purulent discharge, may be symptomatic of a variety of conditions, including the following:

1. *A simple chronic catarrh*, in which the mucous membrane remains more or less congested and slight exposure excites acute coryza.

2. *Chronic hypertrophic rhinitis*, in which there is a spongy overgrowth over the middle and inferior turbinated bones. The surface is covered with thick, tenacious mucus, and nasal spurs and deflections of the septum are common.

3. *Atrophic rhinitis*, in which there is wasting of the tissues which often involves the bone, as a result of which the nasal chamber is enlarged and contains offensive secretions and crusts (*ozæna*).

4. *Caries and necrosis* of the nasal bones is also accompanied by offensive discharge, the nature of the lesion being recognized through the use of the probe to manipulate the sequestrum.

5. *Ulcerations* in the nose are discovered only by examination. They may sometimes be syphilitic, rarely tubercular.

6. *Foreign bodies* in the nose occasion chronic catarrhal symptoms.

Diagnosis.—The recognition and distinction of these lesions is possible only by means of careful anterior and posterior rhinoscopy.

Prognosis.—Simple catarrh is readily curable, and hypertrophic rhinitis becomes so under proper treatment. Atrophic rhinitis, and caries and necrosis, can be mitigated but not cured. The prognosis of an ulceration is governed by its cause. The catarrhal condition due to a foreign body is usually controlled by the removal of the latter.

Treatment.—1. Cleanse and keep clean the nasal cavities. Spray the passages with Dobell's or some similar solution, by means of a nasal douche or an atomizer, until they are cleared of mucus and dust. Follow this with a spray of albolene, plain or mentholated.

2. Remove the hypertrophied tissue. For this purpose the snare, acids, or the galvano-cautery may be used.

3. Improve the general health of the patient. General diathetic states, such as gout or syphilis, should be discovered and treated; and the patient's hygiene, diet, etc., should receive careful attention.

4. Medicines. In simple muco-purulent catarrhs, *hydrastis*, *kali bichromicum*, *pulsatilla*, and *sulphur* are valuable remedies. *Iodide of arsenic* is suited to cachectic patients, *aurum* and *nitric acid* to cases with offensive discharges, and many of the constitutional remedies may be indicated by associated conditions.

HAY FEVER.

(Autumnal Catarrh; Rose Cold; etc.)

Etiology—The *exciting cause* appears to be the inhalation of some irritating substance, such as the pollen of plants. The *predisposing factors* are the spring or autumn season, pre-existing chronic rhinitis, heredity, a neurotic constitution, and the uric acid diathesis.

Symptoms.—The disease is a peculiar catarrhal affection which recurs with marked periodicity once each year. The onset occurs suddenly at the appointed time, the attack beginning with *sneezing*, *coryza*, occlusion of the nostrils, burning and pricking sensations, and profuse watery discharges. Headache is common, conjunctivitis may be present, and *bronchitis* or *asthma* may develop at any time. As the attack progresses the discharge becomes thicker and muco-purulent.

Prognosis.—For relief, good; for permanent cure, doubtful.

Treatment.—The attack promptly disappears upon removal to certain districts, notably the Adirondacks and White Mountains. Temporary relief may be obtained by cleansing the nasal passages with alkaline sprays and applying hydrogen dioxid or solutions of supra-renal extract to the mucous membrane. Permanent cure may be secured in cases due to chronic catarrhs or gout by persistent treatment of the latter conditions. *Arsenic* is the most successful medicine; it may also be used as *potassium arsenite*, *quinine arsenite*, *cuprum arsenite*, or *arsenic iodide*. *Kali iod.*, *sticta*, *euphrasia* and many other remedies may be successful when indicated.

ACUTE CATARRHAL LARYNGITIS.

Etiology.—Acute inflammation of the mucous membrane of the larynx may be caused by exposure to cold and wet, the inhalation of irritants (dust and chemicals), the excessive use of the voice, and traumatism by foreign bodies, corrosive poisons, etc. Heredity, bad hygiene, and diathetic states predispose to it.

Symptoms.—The throat becomes dry and the *voice husky* or lost. Tickling in the throat produces a dry, teasing *cough*, which is later accompanied by viscid expectoration, and there may be some fever and *pain in the throat*, the latter being more marked on speaking or swallowing. In children spasm of the glottis is added, and produces stridulous breathing and croupy cough (*spasmodic*, *catarrhal*, or *false croup*). Examination reveals redness of the mucous membrane, involving the vocal cords.

Diagnosis.—By symptoms and examination.

Prognosis.—In adults the prognosis is favorable unless edema supervenes. In children the danger to life, in spite of the alarming character of the symptoms, is usually slight.

Treatment.—Children subject to attacks of acute laryngitis should spend their lives out of doors, their hygiene and constitutional conditions being carefully investigated in the mean-

time. During an attack the patient should be placed in a room the atmosphere of which is kept at an even temperature (70°) and moist with steam. *Aconite* should be given early, and as the symptoms become more severe *iodine*, *bromine*, or *spongia* may be prescribed. *Belladonna*, *hepar*, *kali bich.*, or *phosphorus* is less often required. Rarely intubation or tracheotomy may be necessary.

CHRONIC CATARRHAL LARYNGITIS.

Etiology.—This is due to the same causes as the acute form.

Symptoms.—Persistent *hoarseness* and *cough*, viscid expectoration, and tickling in the throat. *Examination* reveals congestion of the laryngeal mucous membrane, with small granulations, and reddening of the vocal bands.

Prognosis.—Guardedly favorable under proper treatment.

Treatment.—Complete rest of the voice and the avoidance of exposure are necessary. Spray the naso-pharynx and larynx freely with alkaline solutions, following that with sprays or direct applications of iodine or astringents. *Causticum*, *hepar*, *kali bich.*, and *phosphorus* are the more important medicines.

EDEMATOUS LARYNGITIS.

Etiology.—Edema glottidis may be—

1. Primary, originating in the larynx (rare).
2. Contiguous, spreading from adjacent tissue (pharynx).
3. Consecutive, following perichondritis or other disease of the larynx.
4. Symptomatic of certain diseases, especially the acute infections.

Symptoms.—A feeling of obstruction in the larynx is quickly followed by *dyspnea*, stridulous breathing, and sometimes *suffocation*. The swelling is easily seen with the laryngoscope, often without it.

Treatment.—Place the patient at rest and let him hold in the mouth or swallow small pieces of ice. If the symptoms are threatening, scarification of the laryngeal tissue, intubation, or tracheotomy is advisable. *Apis*, *arsenic*, *iodine*, *lachesis*, and *sanguinaria* are the remedies usually recommended.

TUBERCULAR LARYNGITIS.

Etiology.—As a rule this is secondary to pulmonary tuberculosis; rarely it is primary.

Symptoms.—The early symptoms are identical with those of simple catarrhal laryngitis, including *hoarseness* and *aphonia*. Later *painful deglutition* is complained of, and this, together with the obstinacy of the disease, indicates its true nature. Before that time, however, *examination* should have been made. It reveals either pearly granulations (miliary tubercles) or small, shallow ulcers with gray bases on the pale mucous membrane.

Treatment.—In the hands of a skillful laryngologist applications of lactic acid may be made to the ulcers, or iodoform may be dusted into the larynx. In many cases it is necessary to apply cocain to the larynx just before meals in order to overcome the painful deglutition. The general treatment of tuberculosis must be carried out at the same time.

SYPHILITIC LARYNGITIS.

Symptoms.—In the victim of syphilis, hoarseness, cough and difficult deglutition should insure examination of the larynx. This may reveal an apparently simple laryngitis, or mucous patches, ulcers, and even necrosis of the cartilages.

Treatment.—Local cleansing with sprays, applications of silver nitrate, and active mercurial medication are advisable.

DISEASES OF THE LOWER RESPIRATORY TRACT.

As a rule attention is attracted to disease of the respiratory apparatus (lungs, bronchial tubes, or pleuræ) by symptoms directly referable to those organs, *e. g.*, *chest pain*, *cough*, *expectoration* of mucus or blood, *dyspnea*, and changes in the voice. It must not be forgotten, however, that serious disease may attack the organs of respiration without causing local symptoms, the patient complaining only of *weakness*, *loss of appetite and of flesh*, and perhaps *feverishness*. Because of the insidious nature and vague symptoms of certain diseases, particularly those of tuberculous origin, it is customary to include the respiratory organs in every systematic examination.

Interrogation of the patient.—Should the symptoms elicited by general interrogation point to disease of the bronchi, lungs, or pleuræ, the examiner should inquire particularly regarding:

1. A *family history* of bronchitis, asthma, phthisis, or scrofula.

2. The patient's *occupation:* is he exposed to irritating fumes or dust?

3. *Symptoms.* Has he ever had enlarged glands in the neck? Does he sweat at night? Is he losing weight?

Cough: its character and frequency; when is it worst? Does it cause any pain? Does it ever cause him to vomit?

Expectoration: its amount and character. Is it yellow or not? Does it ever contain blood? If so, is it only after severe coughing? Is the blood dark, or frothy and bright?

Chest pain: is it worse on taking a full breath? Is it constant? What is its exact location?

Dyspnea: when does it occur? If it is paroxysmal, let him describe an attack.

Having satisfied himself as to these details, the examiner should proceed to the physical examination of the patient.

PHYSICAL EXAMINATION OF THE RESPIRATORY ORGANS.

Place the patient, stripped to the waist, in a good light. It is well to inspect the chest with the light striking it obliquely as well as directly. By INSPECTION the examiner determines:

1. **The size, form and nutrition of the chest.**

A. Is the chest *healthy?* (symmetrical, well-nourished, etc.).

B. Is it symmetrical but suggesting proclivity to disease? *e. g.*,

A flat chest or "paralytic thorax," *i. e.*, antero-posterior flattening. This is not necessarily phthisical. There may be emaciation of the chest wall, indicating nutritional failure.

C. Is it symmetrical but suggesting past disease? *e. g.*, the rachitic chest. There may be projection of the sternum ("pigeon-breast"); depression of the root of the sternum ("funnel-breast"); or the "rachitic rosary" (a line of eminences along the chondro-costal articulations, more often felt than seen).

D. Is it symmetrical but suggesting present disease? *e. g.*, the "barrel-chest" (increased antero-posterior diameter) of emphysema. Or

Bilateral retraction of chest walls, *i. e.*, an extremely flat chest.

E. Are there local changes? For example,

Deformities. Spinal curvature may be detected by marking, if necessary, the position of the spinous processes. Such curvature may give rise to considerable displacement of the thoracic organs and so render useless the usual landmarks. Pott's disease may be detected, often better by touch than by inspection; in its early stages it gives rise to muscular rigidity, which must not be confounded

with abnormal rigidity of the spinal column itself, which persists when the patient bends; the latter is due to spondylitis deformans.

Unilateral flattening may be present as the result of chronic phthisis, pulmonary fibrosis, or pleuritic adhesions.

Unilateral prominence may suggest pneumothorax, pleural effusion, or possibly emphysema.

Local bulging may be due to aneurism of the aortic arch, tumor (lipoma, gumma, sarcoma) of the chest wall or viscera, "cold abscess" (tubercular) of rib or sternum, or empyema perforating the thoracic wall. In children, in whom the bony framework is soft, precordial bulging may indicate an enlarged heart or pericardial effusion.

Pallor, cyanosis, edema, jaundice, scars, and eruptions, if present, should be noted and may lead to a correct diagnosis.

Enlarged glands in the neck or axillæ may suggest tuberculosis, syphilis, or malignant disease.

2. **Changes in Respiratory Movement.**

A. *Normal* rate, rhythm, and type.

B. *Abnormal degree of expansion.*

a. Diminished expansion of one side.

If the chest is distended it may be due to pneumothorax, pleuritic effusion, or tumor.

If retracted it is apt to be due to phthisis, pleuritic adhesions, occlusion of a bronchus by an aneurism, etc.

Restriction of motion of one side of the chest may also be due to pressure from enlarged viscera or tumors below the diaphragm.

b. Increased expansion of one side of the chest is usually compensatory to interference with respiration on the other side.

C. *Dyspnea* (difficult breathing) is usually rapid as well as labored, and is often accompanied by cyanosis. It may be inspiratory, as when the larynx is obstructed (croup, foreign body), or expiratory, due to difficulty in emptying the chest (asthma, emphysema).

Simple *rapidity* of respiration, not dyspnea, is a common result of muscular exertion, neurotic disturbances, heart and lung disease; and it usually accompanies fever.

3. **Changes in Respiratory Rhythm** occur in *asthma*, where inspiration is a short gasp and expiration a long wheeze; and in *emphysema*, where, however, less dyspnea is present.

In *Cheyne-Stokes respiration*, periods of apnea (no respiration) are followed by respirations which are first short and shallow, but increase gradually until they are rapid and deep, and then as gradually die away until the patient is apneic again. This respiratory anomaly occurs in connection with severe heart, kidney, or brain diseases and is of grave significance.

Restrained breathing, the patient "catching" his breath, is common in pleurisy.

Shallow and irregular breathing occurs in connection with profound unconsciousness from any cause (apoplexy, uremia, poisoning).

Stridulous breathing, "crowing," is the result of obstruction at the glottis; it occurs in laryngismus stridulus, whooping cough, and larygeal or tracheal obstruction by a foreign body.

4. **Changes in the Diaphragmatic Movements** may be observed by Litten's method. The patient lies on his back with his chest bared and his feet pointing directly to the only window through which light is permitted to enter. As the patient takes a full breath, the observer, standing at his side, can see a narrow shadow which moves down along the axilla from the seventh to the tenth rib. During expiration the shadow, less easily seen, rises again to

its starting point. The phenomenon represents the excursion of the diaphragm during respiration. It is absent in pneumonia of the lower lobe, pleuritic effusion, or extensive adhesions. Fluid or solid tumors below the diaphragm, unless very large, do not abolish it. Hence this procedure may be of great value in distinguishing an enlarged liver or a subphrenic abscess from fluid in the right pleural cavity.

PALPATION, usually practiced in connection with inspection, determines—

1. **The form of the chest,** confirming or modifying the results of inspection.

2. **Vibrations of the chest wall.**

A. *Vocal or tactile fremitus*, the sense of vibration communicated to the hand laid flat upon the chest while the patient speaks (usually counting "one, two, three" or "ninety-nine" repeatedly), varies greatly in different parts of the chest. Allowance being made for these normal disparities, fremitus may be—

a. Diminished or *absent*, when the lung is pushed away from the chest wall by air or fluid in the pleural sac.

b. Increased when the lung is solidified in pneumonia or phthisis.

B. *Pleural friction*, the rubbing together of the roughened surfaces in dry pleurisy, may be felt as well as heard.

C. *Rales*, coarse and dry in character, sometimes communicate a peculiar sensation to the palpating hand.

3. **Tenderness** of the thorax may be found in intercostal neuralgia, in which case it corresponds to the points of exit of the intercostal nerves; and it occurs also in case of necrosis of a rib, in dry pleurisy, and occasionally in phthisis.

4. **Fluctuation** or elasticity of any tumor or prominence in the

chest wall will convey important information as to its character.

5. The **skin temperature;** *e. g.*, the hot, dry skin of fever or the cold, clammy skin of circulatory failure, may be detected by the palpating hands and affords information of diagnostic importance.

PERCUSSION has for its object the determination of two things, viz.,

1. **The boundaries of the lungs** (topographical percussion).
2. **The resonance of the lungs,** including

A. Variations *normal* to various parts of the chest.

B. Variations of resonance *abnormal* in character.

a. Quantitative. Increase (hyper-resonance) indicates emphysema, cavity, etc.

Decrease (varying in degree from slight impairment to absolute dulness) indicates thickening of chest wall, pleuritic effusion, or solidification of lung tissue.

b. Qualitative. Pitch may be low, medium or high; a clear sound of heightened pitch indicates airless structure (consolidation, effusion) behind air-containing structure.

Cracked-pot resonance, a peculiar "chinking" sound produced over a pulmonary cavity or an open pneumothorax from which the air is suddenly and forcibly expelled by the percussion stroke, is best heard by having the patient hold the chest piece of the stethoscope in front of his open mouth, leaving the examiner's hands free to percuss the chest. This sound may be obtained over the chest of any healthy infant, especially when crying.

Amphoric resonance, metallic and echoing in character, may be imitated by percussing the cheek distended with air. It is sometimes obtained in pneumothorax and in phthisical excavation.

AUSCULTATION determines :

1. **The character of the respiratory sounds.**

A. *Vesicular breathing* (breezy in character) is heard normally over lung tissue. Its intensity may be—

a. exaggerated in children.

over thin chest walls.

after exertion leading to deep forcible breathing.

over one lung (compensatory) when the other is out of use (*e. g.*, as the result of solidification or compression by pleural effusion).

b. diminished, as a result of—

the presence of air, solid, or fluid in the pleural cavity.

emphysema, with feeble chest movement.

bronchitis or edema, lessening the inflow of air.

pain in the thorax, because of which the patient restrains the chest movements.

occlusion of the upper air passages (bilateral diminution if the glottis is occluded, unilateral if a bronchus)

paralysis of the muscles of respiration.

B. *Bronchial breathing*, normally heard over the trachea and primary bronchi, is pathologic if heard elsewhere and indicates solidification of that part of the lung (phthisis, pneumonia, etc.), or *distant* bronchial breathing may be heard over a pleural effusion.

C. *Broncho-vesicular breathing*, intermediate in type between the bronchial and the vesicular, is heard normally near, not over, the trachea, and over the larger bronchi at those portions of the chest where they are within auscultatory distance but separated from the chest wall by a small amount of lung tissue. In the healthy chest a broncho-vesicular note is heard over the lower part of the manubrium, over the inter-scapular regions at the

level of the third dorsal vertebra, and over the apex of the right lung. It may be heard elsewhere when disease has produced moderate solidification of the lung.

D. *Amphoric breathing*, comparable to the hollow, empty sound produced by blowing across the top of a bottle, is occasionally heard over a large empty cavity within the chest or over an open pneumothorax.

2. **Adventitious Sounds.**

A. *Rales*, produced by the passage of air through bronchi which contain mucus or pus, or which are narrowed by swollen walls, may be—

Moist or bubbling in character (*crepitations*), and coarse (*large "mucous"* rales), medium, or fine (*subcrepitant*), according to the size of the tube in which they are produced; they indicate the presence of fluid.

Dry or crackling (*rhonchi*), without the bubbling character, because the secretion is thick and tenacious. They are described as large (low-pitched, *sonorous*), medium, or fine (small, *sibilant* rales). The finest of all are the *crepitant* rales, which resemble the sound heard on rubbing a lock of hair between the finger close to the ear, and are present early in pneumonia and more rarely in phthisis, edema, etc.

The groaning, shrieking, and whistling sounds caused by the passage of air through tubes narrowed by swollen membrane, are also described as *musical rales*.

B. *Pleural friction sounds*, rubbing and creaking in character, are heard close to the ear, becoming louder if the pressure of the stethoscope is increased, and, unlike rales, are not influenced by coughing. They suggest the roughness of the inflamed pleural surfaces.

C. *Splashing sounds*, heard if the ear is held against the chest and the patient shaken firmly, indicate the pres-

ence of both air and fluid in a cavity (pyopneumothorax, etc).

D. *Metallic tinkling*, the sound of a drop of liquid falling into fluid at the bottom of a cavity, occurs occasionally in pneumo thorax and less often in large pulmonary cavities.

3. **Vocal Resonance.**

A. The *whispered voice*, the patient whispering "one, two, three" or "ninety-nine," is heard normally only over the trachea and primary bronchi. When audible elsewhere it indicates solidification or excavation of the lung.

B. The *spoken voice* conveys less valuable information than the whisper; it corresponds closely to tactile fremitus in its modification by tissue changes. An increase of vocal resonance is known as *bronchophony*, and the transmission of distinct words rather than confused sound, is called *pectoriloquy*. It is discovered more often in connection with the consolidation of pneumonia than that of phthisis; it may occur also in cases of pneumothorax and pulmonary excavation. *Egophony*, a nasal or squeaky quality of the vocal resonance, may be heard in connection with pleural effusions and occasionally over consolidated lung.

BRONCHITIS.

Bronchitis is a catarrhal inflammation of the bronchial tubes, of which the chief symptoms are cough, expectoration, and dyspnea. Clinically we distinguish:

1. Acute bronchitis of the larger tubes (simple bronchitis).
2. Acute bronchitis of the smaller tubes (capillary bronchitis).
3. Chronic bronchitis.
4. Plastic bronchitis (acute and chronic).

ACUTE SIMPLE BRONCHITIS.

Etiology.—The *exciting causes* of acute bronchitis include—

1. Chill from exposure to cold and damp.
2. Infectious diseases (measles, typhoid, etc.).

The *predisposing causes* include—

1. Impurity of the air (deficient ventilation, fog, dust).
2. Obstruction to the circulation of blood through the lungs (chronic valvular disease, emphysema).
3. Toxemic irritation (gout, Bright's disease).
4. Personal susceptibility (heredity, debility, etc.).

Pathology.—A bilateral catarrh of the larger tubes, usually involving the trachea, is present. The mucous membrane is congested, and there is an increased secretion of mucus mixed with desquamated epithelium and leucocytes.

Symptoms.—The attack may begin as an ordinary catarrh of the nose and throat, thence extending down to the bronchi; or a chill may be followed immediately by the typical symptoms. These are:

1. *Cough*, dry in the early stages and later accompanied by free muco-purulent expectoration.
2. *Pain*, soreness, or rawness under the sternum; when secretion is established, this is relieved.
3. Moderate *fever* (99°–102°) and the usual mild febrile symptoms (headache, coated tongue, etc.).

Physical Signs.—These are often absent. Rales (early, dry; later, moist) may be present.

Diagnosis.—*Influenza* is distinguished by its high fever, intense pains, and great prostration.

Tuberculosis may present only catarrhal symptoms; if the physical signs are limited to one upper lobe or both apices, search should be made for the tubercle bacillus.

The onset of *acute infections*, such as measles or whooping

cough, may be marked by bronchitis, but the later development of typical symptoms and signs will correct the diagnosis.

Congestion of the lung secondary to mitral disease may be deceptive occasionally, owing to the disappearance of the murmur; but careful physical investigation of the heart, together with the probable reappearance of the murmur as the result of rest and treatment, will establish the diagnosis.

Prognosis.—Good, except at the extremes of life, when capillary bronchitis or broncho-pneumonia may develop.

Treatment.—An attack may be aborted at the onset by the use of a hot foot or general bath, followed by rest in bed and the administration of *aconite* or *gelsemium.*

During the dry stage, with painful cough and scanty expectoration, such remedies as *ferrum phos.*, *bryonia*, or *squills* are indicated. Hyperesthesia of the larynx, with tickling cough, suggests *hyoscyamus;* while sensitiveness to external pressure, which excites cough, indicates the need of *rumex*, *sticta*, or *lachesis. Ipecac* and *apomorphia* aid in promoting secretion; and the appearance of free expectoration calls for *antim. iod.* or *kali bich.* Cyanotic symptoms may demand *tartar emetic*, or *antim. arsen.* Co-existing laryngitis may call for such remedies as *phosphorus*, *spongia*, *belladonna*, or *hepar.* In old people difficulty in expectoration is relieved by *senega* or *ammon. carb.* Protracted sub-acute cases may require *pulsat.*, *sulphur*, or *stannum.*

The dry cough of the early stage is often greatly relieved by the inhalation of steam or of nebulized oils.

CAPILLARY BRONCHITIS.

(Suffocative Catarrh.)

Etiology. — The causes are identical with those of milder bronchitis, the more severe development being due to weaker individual resistance (infancy and old age).

Pathology.—Either by extension from the larger tubes, or

primarily, the mucous membrane of the finer tubes becomes inflamed, swollen, and occluded with tenacious mucus. Extension of the disease to the alveoli (broncho-pneumonia) or collapse of the lung areas beyond the bronchial occlusion usually follows.

Symptoms.—1. *Cough* with little expectoration.

2. *Rapid respirations* (68–80 per minute).

3. *High fever* (104°–105°) with restlessness and delirium.

4. *Dyspnea* and *cyanosis*, with weak, rapid pulse (120 or more) and evidence of failing heart.

Physical Signs.—1. Distention of the upper part of chest with recession of the lower, due to respiratory stenosis.

2. Fine "hissing" rales posteriorly on both sides.

3. The ordinary sounds of bronchitis (dry rales) over the upper anterior part of chest.

4. Dulness over the areas of collapse or of broncho-pneumonia.

Diagnosis.—*Pneumonia* has a sudden onset, a severe rigor being followed with high temperature and pain in the side, but there is less subjective dyspnea and usually the physical evidences of pre-existing bronchitis are lacking.

Miliary tuberculosis has an insidious onset, with pallor, wasting, etc.

Prognosis.—This is very grave at the extremes of life, death from heart failure or asphyxia frequently occurring within a few days. In vigorous adults the disease is less fatal.

Treatment.—Place the patient at absolute rest in a room heated to 70° or more and keep the air moistened with steam. Stimulation of the respiratory muscles by gentle friction is helpful. In addition to the other remedies useful in simple bronchitis, grave symptoms, such as dyspnea or cyanosis, call for the administration of *tartar emetic*, *antim. arsen.*, or *apomorphia*. Evidence of impending heart failure demands alcoholic or other stimulants.

CHRONIC BRONCHITIS.

Etiology.—Chronic bronchial catarrh may be:

1. A sequel of repeated acute attacks, and consequently due to the exciting causes of the latter (cold, exposure, etc.).

2. Secondary to local lung lesions (emphysema, phthisis, and pulmonary stasis from mitral disease).

3. Associated with general sclerotic changes (gout, chronic nephritis, senile degeneration).

Pathology.—The mucous membrane is smooth, congested, of greyish hue, and thickened with a connective tissue overgrowth. Irregular dilatation of the tubes (bronchiectasis), emphysema, and dilatation of the right heart often ensue.

Symptoms.—1. *Cough*, not necessarily continuous, but tending to become persistent (*e. g.*, the chronic "*winter cough*" of elderly people).

2. *Expectoration*, which may be scanty (*dry catarrh*), severe paroxysms of cough dislodging little sputum; or profuse and muco-purulent, and sometimes extremely copious (*bronchorrhea*).

3. *Dyspnea*, especially on exertion, in cases of long standing in which emphysema has developed.

Physical Signs.—Moist rales, often associated with feeble breath sounds and other evidences of emphysema.

Diagnosis.—*Tuberculosis* must be excluded by the absence of evidences of consolidation and of the tubercle bacillus in the sputum.

Prognosis.—Under favorable circumstances the bronchial catarrh can be controlled; but emphysema cannot be removed, and associated changes in the heart or kidneys will render the prognosis much more grave.

Treatment.—1. Attention to the cause (gout, heart or kidney disease).

2. Hygienic: nutritious diet, warm clothing, bathing, etc.

3. Climatic: if possible change of residence to a climate affording even temperature, moderate warmth and dry atmosphere.

4. Medicinal. Dry catarrhs are benefited by such remedies as *bellad.*, *iodine*, *rumex*, or *spongia*. With free muco-purulent expectoration the list of useful medicines includes *arsen. iod.*, *antim. iod.*, *stannum*, *stannum iod.*, *kali bichrom.* or *carb.*, *lycopodium*, *pulsatilla*, and *sulphur*. Difficult expectoration in the aged suggests the use of *ammon. carb.* or *senega*. Associated changes in the heart or kidneys will furnish other therapeutic indications.

Inhalations of creosote or terebene are of great value in cases attended with free muco-purulent expectoration.

PLASTIC BRONCHITIS.

(Croupous, Membranous or Fibrinous Bronchitis.)

This is a rare disease of uncertion causation in which the ordinary symptoms of bronchitis are accompanied from time to time with the expectoration of fibrinous casts of the tubes, often with a small quantity of blood. The liability to recurrence of these acute attacks renders the disease essentially chronic.

The prognosis for life is good; for recovery bad, often unfavorable. The treatment is that of chronic bronchitis.

BRONCHIECTASIS.

(Dilatation of the Bronchial Tubes.)

Etiology.—Dilatation of the bronchial tubes is invariably secondary to lesions weakening the bronchial wall and permitting its dilatation under coughing pressure; these include:

1. Congenital weakness of the bronchial wall (rare).

2. Inflammations leading to atrophy of the bronchial wall (chronic bronchitis).

3. Obstruction by foreign bodies or tumors, the tube beyond the point of obstruction becoming dilated.

4. Traction on the tube by the adjacent lung (pulmonary collapse, pneumonia, fibrosis, etc.).

Pathology.—Cylindrical dilatation or sacculation of the tube, occurs, and it is usually associated with local inflammatory or ulcerative changes in the mucous membrane. Fetid secretion accumulates in the sac thus formed, and ultimately occasions characteristic symptoms.

Symptoms.—Moderate dilatation of a bronchial tube causes no symptoms, but the presence of a sacculation gives rise to *paroxysms of cough* attended by the expectoration of large quantities of purulent, often *fetid*, *secretion*. This results from the sac, itself insensitive, becoming gradually filled up and overflowing; with the cough thus excited, the collection is emptied out and for some hours, while the sac is again filling, the patient may be free from cough.

Physical Signs.—As a rule, these are limited to indications of the associated conditions; but in well-marked cases the sacculation affords the physical signs of a cavity (tympany, mephoric breathing, gurgling rales, bronchophony). These are discoverable only when the sac is quite empty. Its location at the base of the lungs aids in distinguishing the latter from a tubercular excavation.

Diagnosis.—*Tuberculosis* is distinguished by the presence of tubercle bacilli and elastic tissue in the sputum; usually there is fever and cachexia.

A circumscribed *empyema* rupturing into the lung presents similar physical signs, but its sudden development, with a history of previous pleurisy, establishes the diagnosis.

Prognosis.—The prognosis as to life is governed by the associated conditions. Bronchiectasis itself is chronic and only palliation is possible.

Treatment should be directed to the causative condition. Emptying of the sac is aided by gravity if the patient "coughs down hill," *i. e.*, lies on a couch with head and shoulders

hanging down toward the floor. Antiseptic inhalations or sprays (creosote, carbolic acid), or intra-tracheal injections of mentholated oil may lessen the fetid expectoration. The drainage of the sac by surgical means may be considered.

ASTHMA.

(Spasmodic, Bronchial, or Essential Asthma.)

Symptomatic dyspnea of varying origin has been mis-called asthma, with a prefix (cardiac, renal) to denote its source. The term should, however, be restricted to the neurotic form.

Etiology.—The attack is due to spasmodic contraction of the bronchioles, of reflex origin, and its causes may be—

1. Neurotic temperament and heredity.
2. The gouty diathesis.
3. Diseases of the respiratory tract (naso-pharyngeal catarrh, adenoids, polypi; enlarged tonsils; bronchitis).
4. Various irritants (certain climates, odors, dust, etc.).

Symptoms.—The typical attack is a *paroxysmal dyspnea* usually occurring at night. After few, if any, prodromes, the patient is suddenly seized with a sense of oppression, and this rapidly develops into distressing difficulty in breathing. Expiration is labored and becomes a prolonged wheeze; the chest is distended and the auxiliary muscles of respiration are called into use without effect. At the height of the attack the face become pale and perhaps cyanosed, and the body is cold and covered with sweat; the patient's distress is frightful. At first there is a tight cough without any rales, but later moist rales are heard, and small, tough masses (*perles*) of sputum are expectorated. These balls can be unrolled in water and appear to be spirals of mucus moulded in the finer tubes (Curschmann's spirals); they contain octahedral crystals (Leyden's) identical with those found in leukemic blood and in semen.

After a period of suffering often extending over several hours, the breathing becomes easier, the cough lessens, and

the patient falls asleep. The attack may recur in a few hours, or the patient may remain well for weeks.

Diagnosis.—*Symptomatic dyspnea*, due to cardiac or renal disease, must be excluded by careful consideration of the history and by an examination directed to those organs.

Laryngeal or *tracheal obstruction* occasions inspiratory, not expiratory, difficulty; and the chest is diminished in size, not distended.

Prognosis.—A cure is rendered possible by discovery and removal of the cause. The disease if uncontrolled tends to produce emphysema and dilatation of the right heart; and these conditions, when established, render recovery impossible, although life may last for years.

Treatment.—The paroxysm may be modified by the administration of such remedies as *arsenic*, *grindelia*, *ipecac*, *lobelia*, and *nux vomica*. If these fail and the attack be extremely violent, recourse may be had to inhalations of *amyl nitrite* or to *morphia*, gr. ¼, hypodermatically; but in general these palliative measures are distinctly prejudicial to ultimate recovery.

In the intervals between paroxysms careful search must be made for the cause. The gouty diathesis if present must be overcome, nasal and pharyngeal diseases must be treated, and the patient's general hygiene should be supervised is every detail.

The remedies which have proven useful include *bryonia* and *ipecac* in cases associated with bronchial catarrh; and *cuprum*, *cuprum arsen.*, *chininum sulph.*, *arsen.*, *nux vomica*, *hydrocyanic acid*, and *chloride of gold* in purely neurotic cases. Other remedies may be indicated by associated conditions.

CONGESTION OF THE LUNGS.

Etiology.—Hyperemia of the lungs may be—

1. Active. It occurs as an early stage in the various inflammatory affections of the lungs, or independently as the

result of inhaling hot air, irritants, etc. It may occasion dyspnea, cough, and expectoration. A *diagnosis* can be made only by exclusion.

2. PASSIVE. (*a*) *Hypostatic.* Feeble heart action, such as occurs in the aged or infirm or the victims of long-continued fever, together with altered blood states and the force of gravity, may lead to congestion of the dependent portions, usually the bases, of both lungs. Following this, the air cells may collapse and a species of pneumonia develop (*hypostatic pneumonia*).

(*b*) *Mechanical or obstructive.* Any condition, such as mitral disease or dilatation of the right ventricle, which prevents the return of blood to the left heart, leads to congestion, followed by connective tissue hyperplasia and pigmentation, in the lung (brown induration). The symptoms are principally those of the heart lesion; they include cough, dyspnea, palpitation, and oppression, and sometimes hemoptysis. The discovery of generally distributed fine rales indicates the pulmonary condition.

3. EDEMA OF THE LUNGS represents a further stage of congestion. Effusion of serous fluid into the air vesicles may occur as:

(*a*) *Collateral edema*, a localized process around an inflamed area.

(*b*) *General pulmonary edema*, due to

(1) Weakness of the left heart, the right remaining unimpaired. In consequence of this the pressure of the blood in the pulmonary circulation becomes increased to such an extent that exudation of serum into the air vesicles ensues.

(2) Hydremic conditions associated with impaired nutrition of the vascular walls, such as are present in cases of profound anemia nephritis, septicemia, etc., may lean to escape of the serum into the vesicles. This often occurs during the death agony.

Symptoms.—The onset of pulmonary edema is attended with an increased frequency of respiration, dyspnea, and cough; and the latter is accompanied by the expectoration of a frothy, watery, and sometimes blood-stained, serum. Fever does not occur unless there is an inflammatory condition present elsewhere. The extremities are usually cool, and cyanosis may develop. Physical examination reveals incomplete dulness and crackling and bubbling rales over the areas involved, which are usually at the bases of both lungs.

Treatment.—The treatment of congestion of the lungs, whatever its variety, must be directed to the causal condition. In those forms which are associated with heart weakness, and especially if pulmonary edema occurs, it may be necessary to resort temporarily to such remedies as *strychnine*, gr. $\frac{1}{60}$–$\frac{1}{30}$, *caffein sodio-benzoate*, gr. i, or *atropine*, gr. $\frac{1}{100}$. Edema also affords indications for the administration of such medicines as *ammonium carb.*, *arsenic*, *phosphorus*, and *tartar emetic*.

HEMOPTYSIS.

Etiology.—Blood may be expectorated as the result of—

1. Pulmonary lesions.
   (*a*) Pulmonary congestion in the early stages of pneumonia, or secondary to heart disease, etc.
   (*b*) Pulmonary embolism.
   (*c*) Pulmonary tuberculosis.
   (*d*) Pulmonary gangrene.
   (*e*) Pulmonary carcinoma.
2. Bronchial lesions.
   (*a*) Bronchitis, acute, chronic, or fibrinous.
   (*b*) Bronchiectasis.
   (*c*) Ulceration of the bronchial tubes, or of the larynx or trachea.
3. Cardio-vascular lesions, occurring in:

Arterio-sclerosis, aneurism, purpura, hemophilia, and certain acute infections.

4. Traumatism. Injuries and contusions of the chest, etc.

Symptoms and Diagnosis.—As a rule the patient first notices a salty taste in the mouth, and this is quickly followed by the expectoration of frothy, bright red blood in varying quantities. There may be pain in the side, dyspnea, and even syncope. Auscultation may reveal moist rales together with evidences of the causative disease, but any more extended physical investigation is inadvisable until after the hemorrhage has ceased. It is important to exclude *epistaxis*, in connection with which blood may enter the naso-pharynx and be expectorated; and *bleeding from the gums*, or *from the pharynx*. As a rule this can be accomplished by simple inspection. In *hematemesis* the blood is dark in color, often clotted or mixed with remnants of food, and the associated symptoms are referable to the stomach.

Treatment.—Place the patient at rest in a semi-recumbent position, let him swallow small bits of ice, and administer such remedies as *aconite*, *arnica*, *erigeron*, *ergot*, *ferrum phos.*, *geranium*, *hamamelis*, *hydrastinine hydrochlorate*, *ipecac*, and *millefolium*. In grave cases *morphia* may be given hypodermatically.

EMPHYSEMA.

Interlobular or Interstitial Emphysema, the condition in which air, escaping from ruptured vesicles, collects in the interlobular tissue and there appears like rows of beads outlining the lobules, is not recognizable clinically and, moreover, possesses nothing in common with vesicular emphysema but the name.

Vesicular emphysema may be local (compensatory) or general; and the latter may be hypertrophic, "large-lunged," or atrophic, "small-lunged."

Etiology.—The condition is one of dilatation of the alveoli

associated with imperfect contractility of the lung tissue, the loss of elasticity being due to two factors, viz.,

1. Strain of the pulmonary tissue. Certain occupations, such as glass-blowing or performing on wind instruments, and certain diseases, notably asthma and chronic bronchitis, lead to such stretching of the lung tissue that the power of subsequent contraction becomes destroyed.

2. Weakness of the elastic framework, which is either hereditary or acquired.

Pathology.—In the *hypertrophic* form the alveoli become over-distended and this leads to atrophy of their walls, and in connection with the latter their blood vessels are destroyed. The lungs become so distended that when the thorax is opened, they fail to collapse and may even bulge forward.

The *atrophic form* is in reality nothing more than a senile change involving the lungs among other organs. By a process of wasting some of the septa disappear, so that neighboring vesicles become fused into bullae of various sizes. On opening the chest the lungs appear small, pigmented, dry, and easily compressed.

Associated Lesions.—Chronic bronchitis, hypertrophy and dilatation of the right ventricle, and finally chronic venous congestion of the abdominal viscera may ensue.

Symptoms.—1. *Dyspnea*, at first slight and brought on only by exertion, but which finally becomes constant.

2. *Cyanosis*, due to the unærated condition of the blood. In the earlier period of the disease the face may appear congested or slightly bluish, but later in the course cyanosis is marked.

3. *Cough* and *expectoration*, due to the associated bronchitis.

Physical Signs.—In hypertrophic cases the chest may be "barrel-shaped." The more important signs, however, are diminished fremitus, exaggerated percussion-resonance, decreased vocal resonance, and a low-pitched expiratory note which is prolonged, even to three or four times the length of inspiration, and is often accompanied by rales.

Prognosis.—The disease itself is incurable; but the dangers lie in its associations and complications, and much can be done to prevent the progress of the latter.

Treatment.—These patients require a nourishing diet, moderate open-air exercise, and protection from cold and damp. Compressed air inhalations are useful, and medicinal measures should be adapted to the associated conditions (see chronic bronchitis, asthma, and gastric and cardiac diseases).

BRONCHO-PNEUMONIA.

(Catarrhal Pneumonia; Lobular Pneumonia.)

Etiology.—This circumscribed inflammation of the lung tissue is usually a sequence of bronchitis, various septic organisms (staphylococci, streptococci, pneumococci, etc.) being carried into the air vesicles by—

1. Extension of the catarrhal inflammation from the smaller tubes, by contiguity of structure, into the alveoli surrounding them.

2. Sucking into the alveoli of mucus, pus, food products, and other irritants (*aspiration pneumonia*).

Broncho-pneumonia occurs principally in very young children, and occasionally in the aged, but rarely in middle life. It is often secondary to infectious fevers.

Pathology.—The membrane lining the terminal bronchioles becomes swollen, and congested, and the tubes filled with tenacious mucus (*capillary bronchitis*). In addition, the adjacent alveoli are packed with pus cells and epithelium, but fibrin, which is characteristic of croupous pneumonia, is absent. The air vesicles of portions of, or entire, lobules in both lungs may thus become consolidated, while other air vesicles, because of occlusion of their bronchi, are collapsed (*atelectasis*).

On section the lung appears congested, and scattered through the tissue can be seen rounded areas of consolidation

about the size of peas. About these are patches of collapsed lung-tissue and on pressure muco-pus issues from the inflamed smaller bronchi.

Symptoms.—The symptoms, which are usually added to those of a pre-existing disease, include :

1. *Increased frequency of respiration* (50–80 per minute), rapid pulse (140–160), and irregular fever (101°–105°).

2. *Dyspnea* and *cyanosis* are marked. The extraordinary muscles of respiration are brought into play, but the lips and face become cyanosed and the patient may sink into a stupor.

3. *Cough*, which may have been loose and painless previously, becomes dry, hacking, and ineffective. The expectoration is muco-purulent and scanty, or it may be entirely absent.

4. Cerebral symptoms, such as delirium and convulsions, may by their occurrence at the outset of some cases suggest a meningitis, but in a few days they give place to the typical pulmonary symptoms.

Physical Signs.—Scattered points of consolidation, if found, are distinctive; but as a rule the solidified areas are too small to give rise to percussion-dulness, increased fremitus, etc. Auscultation may reveal patches of fine rales, not limited to inspiration, which are associated with the larger moist rales of bronchitis.

Course.—The disease may cause death from exhaustion or asphyxia within a few days. If it does not, the course is usually protracted over two or three weeks and the disease finally terminates by lysis. Chronic interstitial pneumonia or tuberculosis may ensue.

Diagnosis.—The diagnosis often rests upon the symptoms rather than the physical signs, the severity of the former indicating a process much more serious than simple bronchitis.

Prognosis.—This is grave in all well-marked cases, the mortality exceeding 30 per cent. A high temperature and feeble pulse are ominous signs.

Treatment. — These patients require absolute rest, highly nutritious liquid food, and early stimulation with some alcoholic. Respiratory failure should be combated by frictions or cold douches applied to the chest. *Aconite*, *gelsemium*, and *ferrum phos.* are valuable remedies in the early stage, but increasing cyanosis requires the administration of *tartar emetic*, or *apomorphia*. *Phosphorus* and *sulphur* may be suggested by the presence of widespread consolidation. The patient should remain under treatment until it is certain that resolution is complete, lest tubercular infection of an unresolved area occur.

GANGRENE OF THE LUNGS.

Etiology.—Pulmonary gangrene may be either circumscribed or diffuse. It is due to the organisms of putrefaction (staphylococci, leptothrix, etc.), whose activity may be secondary to:

1. Lesions of the bronchi (putrid bronchitis), aspirated foreign bodies, etc.

2. Lesions of the lungs; *i. e.*, as a sequence of some pneumonias and of tuberculosis.

3. Lesions of the blood vessels (septic embolism or thrombosis).

4. Lesions of adjacent viscera (extension of a septic or gangrenous process from the thoracic wall, abdomen, esophagus, etc.).

5. Wounds of the thorax, extravasation of blood into the lung tissue being followed by gangrene.

6. Certain general conditions, such as starvation, alcoholism, diabetes, nephritis, and infective fevers have been occasionally followed by gangrene.

Symptoms.—Cough, dyspnea, moderate fever, and great prostration are present, and are accompanied by a profuse and extremely *offensive expectoration*. The latter settles into layers on standing; it may present a variety of colors, decom-

posed blood giving it a "prune-juice" character; and it contains pus corpuscles, elastic tissue, crystals, and micro-organisms. A more or less profuse hemoptysis may occur.

Physical Signs.—These depend upon the lesions and may indicate consolidation, softening, or excavation; they are not characteristic.

Diagnosis.—By the offensive odor and other characteristics of the sputum.

Prognosis.—This is generally unfavorable, death occurring within two weeks. Rarely the course is more prolonged, and occasionally the slough separates and is discharged, the resulting cavity undergoes contraction, and recovery ensues.

Treatment.—Conserve the patient's strength by administering nourishing liquid foods and alcoholic stimulants. Use sprays or inhalations of creosote, carbolic acid, or terebene; and internally give *arsenic*, *lachesis*, etc.

ABSCESS OF THE LUNG.

Etiology.—Suppuration within the lung may be due to:

1. Pneumonia, the consolidated area undergoing softening.
2. Tuberculosis, a caseous area becoming purulent.
3. Bronchiectasis, ulceration involving adjoining tissue.
4. Pyemic infarction or embolism.
5. Suppuration of a hydatid cyst.
6. Perforation of the lung by a malignant growth of the esophagus, by rupture of an empyema, or by an abscess of the mediastinum, of the bronchial glands, or of the abdomen.
7. Injuries of the lung (wounds, etc.).

The symptoms may be limited to—

Symptoms.—1. *Fever* of hectic type.

2. Sudden *expectoration of pus* in large quantities.

Physical Signs.—Examination may reveal an area of dulness,

which may possibly simulate a pleural effusion; after expectoration of the pus, the signs of a cavity appear.

Prognosis.—Grave, but not necessarily fatal.

Treatment.—Nourish and stimulate the patient, conserve his strength and use antiseptic inhalations as in cases of pulmonary tuberculosis. Consider the possibility of surgical interference and drainage.

TUMORS OF THE LUNG.

With few exceptions, a tumor of the lung, whether carcinoma, sarcoma, or other variety, is secondary to a similar growth elsewhere. As a result it usually consists of multiple nodules which do not attain the size and appearance of neoplasms of other organs. By involving the lung tissue a tumor may occasion *cough*, *hemoptysis*, and *expectoration* (often "prune-juice"); by pressure it may cause *dyspnea*, perhaps *dysphagia*, and vascular obstruction; and through invasion of the pleura *pain* may become severe. The increasing *cachexia* and the discovery of primary or secondary growths elsewhere may enable the physician to make a diagnosis. Treatment must be symptomatic and palliative.

COLLAPSE OF THE LUNG.

(Atelectasis.)

Etiology.—Absence of air from a portion of the lung tissue may be:

1. *Congenital*, respiration never having been sufficiently deep to inflate certain portions of the lungs.
2. *Acquired*, collapse having followed—
   (*a*) Occlusion of the upper air passages by chronic catarrh, enlarged tonsils, adenoids, laryngeal stenosis, etc., which impedes the entrance of air and so limits the expansion of the chest.

(*b*) Occlusion of a bronchus by a foreign body, or a mucous plug, as in broncho-pneumonia.

(*c*) Impaired chest expansion, due to muscular paralysis or weakness.

(*d*) Compression of the lung tissue by a pleural effusion, aneurism, tumor, etc.

(*e*) Pneumo-thorax. Entrance of air into the pleural cavity invariably causes lung collapse.

Symptoms.—Congenital atelectasis is associated with weakness, arrested growth, and symptoms of defective oxygenation, such as shallow respiration, dyspnea, and tendency to cyanosis.

When lung collapse is the result of respiratory stenosis, the onset is apt to be sudden and marked by dyspnea, rapid breathing, and feeble pulse, together with inspiratory recession of the lower intercostal spaces. Extensive collapse may give rise to dulness, and the breath sounds may be feeble or absent over the area of lung involved.

Prognosis.—The degree of cyanosis is the best guide as to the gravity of the condition; if it is increasing, the outlook is unfavorable. If recovery occurs, certain areas may remain unexpanded, and this condition favors the development of bronchiectasis or tuberculosis.

Treatment.—In a newly-born child expansion of the lungs may be favored by a general hot bath, by pouring cold water over the chest, by slapping the chest with a wet towel, or by bringing about artificial respiration. Rather similar methods are of value in the cases of pulmonary collapse attendant upon acute disease, notably in broncho-pneumonia. Later, attention must be paid to the general nutrition, and the patient should take respiratory exercises in the open air.

PLEURISY.

Etiology.—Inflammation of the pleura may be—

1. Primary; due to bacterial infection (pneumococcus, streptococcus, staphylococcus, tubercle bacillus, etc.).

2. Secondary to

   a. Local disease (extension of inflammation from the lungs or injury to chest wall).

   b. General diseases (nephritis, hepatic cirrhosis, cancer, etc.).

Exposure to cold may induce pleurisy by lessening the resistance of the individual.

Pathology.—1. *Dry stage.* The pleural membrane becomes hyperemic and its surface covered with fibrinous exudate. (*Dry*, *fibrinous*, *plastic*, or *adhesive pleurisy.*)

2. *Exudative stage.* Should the inflammatory process continue, a clear, straw colored serous fluid may be poured out, and in the latter are flakes of fibrin (*sero-fibrinous* or *exudative pleurisy; pleurisy with effusion*).

3. *Purulent stage.* In time, or perhaps almost at the onset, pyogenic bacteria may convert the exudate into pus (*purulent pleurisy; empyema.*).

4. *Chronic stage.* Any pleuritic inflammation, plastic or exudative, which remains unresolved after several weeks, constitutes a chronic pleurisy.

The effect of pleurisy upon the adjacent viscera depends upon the quantity of the effusion; if the latter is considerable, the adjacent pulmonary tissue becomes compressed, the heart is displaced toward the sound side, and if the effusion is on the right side the liver is displaced downward.

Course.—1. Resolution and absorption may occur at any stage, although this becomes less common with increasing exudation, and is rare in empyema.

2. Dry pleurisy may occasion permanent adhesion of the surfaces.

3. Unabsorbed serous effusions may remain unaltered for months, but they are more apt to become purulent.

4. Small accumulations of serum or pus may be walled in by adhesions.

5. Purulent effusions, if not promptly removed, may—

a. Perforate the chest wall externally.

b. Burrow along the muscles and point in the lumbar region or groin.

c. Rupture into the esophagus or pericardium.

d. Penetrate the diaphragm, and either enter the stomach or form a pus-collection between the diaphragm and the liver (*sub-diaphragmatic abscess*).

e. Perforate the lung, the pus being discharged through the bronchi and air being permitted to enter the pleural sac (*pyo-pneumothorax*).

Symptoms.—Certain cases of pleurisy are insidious in their development, affording only vague chest symptoms, and in consequence they are detectable only by physical examination. As a rule, however, the onset of acute pleurisy is attended with:

1. *Chill* or chilliness.

2. *Pain*, severe and cutting, in the side; this is increased by any movement, even coughing or breathing, and in order to avoid it the patient breathes in short gasps. When the pleural surfaces become separated by an effusion, the pain lessens and disappears.

3. *Cough*, dry or with scanty expectoration.

4. Moderate *fever* (101°–103°) and a rapid pulse (100–120). Subsequently the fever declines by lysis.

Physical Signs.—1. DRY PLEURISY is suggested by the obviously restricted motion of the affected side, on which the patient usually lies in order to further restrain its movement. Percussion in this stage is negative; but auscultation reveals a grating or rubbing sound, most marked toward the close of inspiration and intensified by pressure of the stethoscope.

2. PLEURITIC EFFUSION, whether serous or purulent, may

obliterate the interspaces, restraining movement and lessening vocal fremitus. In addition, percussion demonstrates:

a. Flatness over the fluid, with a sense of resistance to the finger during percussion.

b. Tympanitic resonance above the level of the fluid.

c. The upper level of the fluid is rarely horizontal; rather S-shaped.

d. The fluid may change its location slowly with a change in the patient's position.

Auscultation may reveal:

a. Feeble or absent vocal and breath sounds over the fluid.

b. Egophony, at the angle of the scapula, if the amount of fluid is small.

c. Bronchial or broncho-vesicular breathing over the condensed lung above the fluid.

d. Exaggerated breathing on the sound side.

e. Displaced heart sounds.

3. During the *stage of absorption* there may be:

a. Gradual disappearance of the dulness.

b. Gradual reappearance of the voice and breath sounds and of fremitus.

c. Pleural friction may recur (*redux friction*).

Diagnosis.—Pleurisy can be diagnosed with certainty only by recognition of its physical signs. The nature of the extravasated fluid can be determined only by exploratory puncture, although a marked and increasing leucocytosis suggests purulency.

Lobar pneumonia may present dulness and bronchial breathing, but vocal fremitus and resonance are increased. Pleurisy is also distinguished by its upper line of dulness, the shifting of the latter when the patient changes his position, and the displacement of adjacent organs. Any doubt may be removed by puncturing the chest wall with a sterile needle and withdrawing a little fluid.

Intercostal neuralgia and *myalgia* are usually distinguished by absence of fever as well as physical signs. The pain of the former is confined to one interspace, along which sensitive foci can be discovered.

Prognosis.—Sudden death from thrombosis or embolism is possible. Excluding this contingency, the prognosis is governed by the variety of the causative organism, the amount and character of the effusion, and the presence or absence of complications.

1. The *exciting cause.* A large proportion of pleural effusions is due to the tubercle bacillus; these cases are often latent and little inclined to purulency. In connection with every case of pleurisy it is advisable to examine the lungs, and especially the opposite apex, for signs of tuberculous consolidation. Pneumococcic infections tend to the production of a mild form of empyema; and this organism is the exciting cause in a majority of the cases in children. Streptococcic infections, on the other hand, are severe, and the death rate is high (25%); these bacteria are responsible for the majority of the cases of empyema which occur in adults, often as a complication of other infectious diseases. It follows, therefore, that careful macro- and microscopic examination of the effusion, together with inoculation tests, is a great aid to definite prognosis.

2. The *amount and character of the effusion.* Pleurisy without effusion tends to recover within a week or two. Should effusion occur, its tendency to become purulent varies according to the exciting cause, as stated. The course of empyema is, at best, protracted.

3. The *occurrence of complications*, especially of pericarditis or meningitis, is of grave import. In secondary pleurisies the prognosis may vary according to the antecedent cause (*e. g.*, nephritis, cancer, etc.).

Treatment.—DRY STAGE. Place the patient at rest. If pain is severe, strap the chest. Use strips of adhesive plaster three inches wide; apply them at the close of expiration, carrying

each from the sound side of the vertebræ around the affected side to the sound side of the sternum. Begin below, and allow the upper to overlap the lower strips slightly.

Administer *bryonia* or *asclepias* in uncomplicated cases. Associated pneumonia or phthisis may require *ferrum phos.*, *kali carb.*, *phosphorus*, or *squills*. A rheumatic cause may suggest the employment of *colchicum* or *rhus;* and nephritis may afford indications for *arsenic*, *canth.*, or *merc. corr.*

Stage of effusion. The lessening of pain as a result of the development of an effusion suggests a change to such remedies as *apis*, *arsenic*, *cantharis*, or *sulphur*. Should, however, the fluid fail to become absorbed after active symptoms have disappeared, or should the distended pleura produce threatening dyspnea or other symptoms, thoracentesis becomes imperative. Aspirate with antiseptic precautions at the upper border of the seventh rib in the mid-axillary line.

Empyema. In purulent pleurisies of pneumococcal origin, thoracentesis may be repeated several times with a fair prospect of recovery without resort to more radical measures. Should the condition be of streptococcic or doubtful origin, however, immediate resection of a rib and drainage of the sac is advisable. After withdrawal of the pus, *arsenic iod.*, *chininum ars.*, *hepar*, *mercur.*, *silica*, and *sulphur* may be useful. Following the evacuation of the pus, healing occurs by obliteration of the sac. In order to expand the collapsed lung tissue, vocal gymnastic exercises, such as blowing water from one large bottle to another, or the inhalation of compressed air, may be advised.

PNEUMOTHORAX.

Etiology.—Air may enter the pleural sac as the result of—

1. *Perforation of the chest wall* by wounds, by incision for drainage of an empyema, or by external rupture of the latter.

2. *Perforation of the pulmonic pleura* by a ruptured tubercular cavity (90%), abscess, gangrene, empyema, rupture of the lung by violent strain, etc.

3. *Perforation of other organs* which contain air, such as the esophagus, stomach, or colon, by an empyema.

Pathology.—The entrance of air destroys the pleural vacuum and the lung shrinks by reason of its own elasticity. The adjacent viscera are displaced. Plural effusion soon develops, and according to its nature the condition becomes a hydro-pneumothorax or a pyo-pneumothorax.

Symptoms.—As a rule there is a sudden onset of *sharp pain* in the chest or back, and a sensation of "something having given way." This is quickly followed by *dyspnea* and perhaps *collapse*. The very acute symptoms, if not fatal, pass off in a few hours.

Physical Signs.—*Inspection.* The affected side is motionless, perhaps obviously enlarged, and the intercostal spaces are obliterated and the apex beat displaced.

Palpation.—Fremitus is absent over the affected side. The heart and liver are displaced.

Percussion.—Loud tympanitic resonance is present over the affected side.

Auscultation.—The respiratory and vocal sounds are inaudible in the lower part of the chest. At the upper part of the chest there is faint amphoric breathing, and cough is followed by a ringing after-echo. If a coin placed on the back of the chest be struck with another coin, the examiner, listening in front, detects a ringing (bell) sound. The "falling drop sound" and "metallic tinkle" are sometimes present.

Diagnosis.—By the physical signs. A large *tubercular cavity* usually occurs at the apex, not the base of the lung; and in such a case the chest wall is sunken, not bulging, the heart is not displaced, and the bell sound is absent.

Treatment.—At the outset administer stimulants if necessary; then strap the chest, and treat the condition simply as a pleurisy with effusion.

HYDROTHORAX.

Etiology.—A dropsical effusion into the pleural cavity is usually secondary to the same causes as is dropsy elsewhere, *i. e.*, to heart, kidney, or blood diseases.

Symptoms.—Dyspnea, cyanosis, and the physical signs of pleural effusion in both sides.

Diagnosis.—By the history of a primary disease, the absence of pain or fever, the bilateral effusion, and on exploratory puncture the withdrawal of a thin fluid, low in specific gravity and poor in albumin.

Treatment.—Treatment should be directed to the causal disease. If dyspnea becomes extreme, aspirate, as in cases of pleurisy with effusion

HEMOTHORAX.

Etiology.—Blood may be effused into the pleural cavity as the result of traumatism, aneurismal or venous rupture, and certain diathetic conditions, such as scurvy and purpura.

Symptoms.—The symptoms are those of hemorrhage—syncope, pallor, etc.—and are severe in proportion to the amount of blood extravasated. Dyspnea and the physical signs of pleural effusion are present.

Prognosis.—This depends upon the cause.

Treatment.—The treatment should be symptomatic unless the dyspnea becomes urgent; in that case a limited amount of the effusion may be withdrawn.

DISEASES OF THE URINARY SYSTEM.

The chief symptoms of disease of the urinary organs may appear at first sight to have no relation to that system. In the investigation of these disorders it is necessary, therefore, that the pathological association of the various organs be kept in mind. The symptoms suggestive of urinary disease are of three classes, viz.:

1. Symptoms, painful or otherwise, referred to the urinary tract (lumbar aching, pain, frequency or difficulty in micturition, etc.).

2. Symptoms due to faulty renal elimination; *i. e.*, uremia (head-ache, vertigo, vomiting or purging, convulsions, coma, etc.) or dropsy.

3. Changes in the appearance of the urine (high colored, smoky, bloody, or very pale; scanty or profuse; containing various sediments, etc.).

In a certain proportion of cases the patient is induced by these symptoms to seek a physician; but more often the indications of a renal disorder remain vague and unnoticed. The effects of chronic nephritis in particular are so far reaching, producing disturbances of the circulatory, respiratory, digestive or nervous systems, that it is the rule to *investigate the urine repeatedly in every case of persistent ill health.*

Interrogation of the Patient.—Should suspicion be directed to the urinary system, the patient should be questioned as to :

1. A *family history* of gout, paralysis, heart, or kidney disease.

2. A *personal history* of acute infections, syphilis, lead poisoning, gout, prolonged suppuration, or previous renal disease.

3. Has he any pain in the lumbar region? Any pain in micturition? If so, is it before, during, or after urination? What is its character and where is it felt? Is it aggravated by movement? Was there ever a sudden attack of pain shooting down into the groin?

Does he have to arise at night to micturate? Is there increased frequency of micturition?

Is the face ever puffy in the morning, particularly beneath the eyes? Do the ankles swell?

4. Does he complain of drowsiness, headache, vomiting, diarrhea, dyspnea, dimness of vision, convulsions, or paralysis? Inquire also as to the condition of the digestive system.

5. The *urine:* is it altered in quantity? In color? Is it clear or cloudy when passed? Is there ever any blood in it, and if so, when?

Physical Examination of the Kidney.—In order to palpate the kidney, sit by the patient on the side to be examined, and place one hand below the last rib behind and the other over the lower part of the hypochondriac region in front. Let the patient's knees be drawn up and his shoulders raised. Then ask him to draw a long breath, and during inspiration follow down the receding abdominal wall with the hand in front. If the patient is not too fat, the lower part of the kidney can thus be felt even in health.

The kidney moves very slightly with respiration. Should it be freely movable in almost any direction, it constitutes a "floating kidney." A distended gall-bladder may be mistaken for the kidney, but the former when pushed back from the abdominal wall springs forward again, while the kidney often disappears for a time, and is seized with difficulty. Moreover, the kidney can be pushed down towards the pelvis and held there, but the gall-bladder moves up again during expiration.

An enlarged left kidney might be mistaken for the spleen,

but the edge of the latter is sharp while the kidney is always rounded. Often the colon is felt as a soft yielding structure in front of the kidney, never in front of the spleen.

Enlargement of the kidney, due to pyelitis, hydronephrosis, tuberculosis, a malignant neoplasm, a cyst, or an abscess, may be palpable. Fluctuation in the mass suggests hydronephrosis or a cyst, while hardness and nodulation signify malignant disease.

Only extreme enlargement of the kidney can produce bulging of the abdominal wall sufficient to catch the eye on inspection. Percussion is much less valuable than palpation.

EXAMINATION OF THE URINE.

By examination of the urine we obtain evidence as to:

1. The *progress of metabolism.* For example, urea, the result of albuminoid disintegration, is excreted in the urine, and by determining its amount we can estimate the relation of nitrogenous expenditure to income. Sugar, appearing in the urine as a result of abnormal metabolism, affords positive evidence of certain grave pathological conditions.

2. The *condition of the kidneys and bladder.* Albumin and certain histologic elements, absent in normal urine, appear in cases of kidney disease. Disease of the bladder may occasion urinary decomposition.

3. The *condition of other organs.* Diseases of the liver may occasion the escape into the blood and thence into the urine of biliary coloring matter. Diseases of the small intestine are frequently accompanied by indicanuria.

The *method of collecting the urine* for examination is important. If possible, the patient should preserve the entire quantity passed during a period of twenty-four hours in a clean, closed vessel (preferably a large, well-corked bottle), and keep it in a cool place to prevent decomposition. At the end of the period the quantity should be accurately measured in ounces or cubic centimetres, and about four ounces should

be brought to the physician for examination. If the microscopical examination cannot be made at once, a few drops of formalin or a few grains of salicylic acid may be added as a preservative; but this may invalidate chemical tests.

PHYSICAL EXAMINATION OF THE URINE.

1. **Quantity.**—This may be modified by the sex or bulk of the patient, the quantity of food and drink, and the amount of excretion by the skin, bowels, etc. The average quantity is about three pints in twenty-four hours; less than one or more than five pints usually indicates disease. The quantity is *decreased* as a result of free sweating, diarrhea, the gouty diathesis, and acute fevers, and in acute and some forms of chronic nephritis. It is *increased* in diabetes, chronic interstitial nephritis, and hysteria.

2. **Color.**—The normal color is yellow. The smaller the quantity the greater the concentration, and therefore the deeper the color. It is *brown*, with yellow foam, when it contains biliary coloring matter; when it contains blood it may be *red*, or intimate admixture of the latter may render it *smoky;* and poisoning with carbolic acid or creosote may render it *black*. Very *pale* urine is found when the quantity is excessive, as in diabetes or chronic interstitial nephritis; and it may also occur in hysteria or as a result of excessive drinking.

3. **Reaction.**—The chemical reaction is ascertained by means of litmus paper. Normally it is *acid*. If it is sharply so, intensely reddening the blue litmus, the possibility of sugar is suggested. Should it be *alkaline*, the red litmus becoming blue, notice whether the red color returns on drying the paper; this indicates a volatile alkali, and if the urine be freshly voided suggests a chronic inflammatory condition of the lower urinary tract. If, however, the blue color persists on drying, the alkali is fixed and indicates unusual alkalinity of the blood, which may possibly be due to fasting, dyspepsia, a vegetable diet, or to alkaline waters or medicines.

4. **Specific Gravity.**—This is taken by means of a urinometer. Normally it is between 1015 and 1025. A very *low* specific gravity is found in all cases where the quantity of urine is large, and in diabetes insipidus, amyloid kidney, and chronic interstitial nephritis. It is *high* in the concentrated urine of fever, the scanty urine of renal or cardiac dropsy, or as a result of a limited consumption of fluids; and it is especially high, and at the same time lncreased in quantity, in cases of diabetes mellitus.

The proportion of solids in any given specimen of urine may be calculated by multiplying the last two figures of the specific gravity by 2.2 (Loebish's coefficient). The result indicates the amount of solids in every 1000 parts.

5. **Cloudiness.**—Normal urine is quite clear. Turbidity in any degree is abnormal, its significance being judged according to the chemical reaction present. Cloudiness in acid urine may be due to urates, which will be dissipated on heating in a test-tube, or to histological elements, which must be detected microscopically. Cloudiness in alkaline urine may be due to the presence of phosphates, rarely calcium oxalate, and will then disappear on the addition of a few drops of acid. Should it persist, it is due to organic matters requiring microscopical examination for their detection.

A milky turbidity of the urine may be due to the presence of chyle (*chyluria*), and in that case after the urine has been allowed to stand for a time a light coagulum settles to the bottom and a creamy pellicle of fat rises to the surface. This condition is the result of some communication between the lymphatic system and the urinary tract. While rare in this climate, chyluria is not uncommon in the tropics, and is there associated with the presence of the filaria sanguinis hominis.

CHEMICAL EXAMINATION OF THE URINE.

The routine chemical examination of urine implies estimation of the amount of urea in the twenty-four hours's urine, and qualitative tests for albumin and sugar. Examination for other substances is reserved for special contingencies.

NORMAL CONSTITUENTS.

1. **Urea,** the chief end-product of albuminoid disintegration, is by far the most important organic element dissolved in the urine. Only by the amount of urea excreted in each period of twenty-four hours can the physician judge as to the extent to which the kidneys are performing their function. Normally the amount ranges from 300 to 600 grains (20 to 40 grammes) daily in a man of average weight (150 pounds). Its persistent diminution below 250 grains strongly suggests kidney disease, and if it falls below 175 grains daily, uremia is to be apprehended. It is decreased also in acute yellow atrophy of the liver. It is increased in fever, anemia, and poisoning by arsenic and other drugs.

For clinical purposes the best test is that with a sodium hypobromite solution, using Doremus' ureometer. To ten c.c. of a caustic soda solution (3 ounces of caustic soda in 8 ounces of distilled water) add 1 c.c. of bromine, and mix thoroughly; then add an equal quantity of water, and fill the bulb of the instrument with the mixture, inclining it until the solution fills the long arm. Into the latter slowly discharge 1 c.c. of the urine. Immediately nitrogen gas is evolved as the result of decomposition of the urea and collects at the top of the tube. When no further evolution of gas occurs, read off the quantity as marked by the numbers on the tube, each of which repreents the fractions of a gramme of urea per c.c. of urine, of which 0.02 is about normal; multiplying the figure by 100 gives the percentage of urea. From this the total daily excretion can easily be reckoned.

A mixture of 25 c.c. of Labarraque's solution with 5 c.c. of a 20 per cent. potassium bromide solution may be substituted for the usual hypobromite solution, but the reaction, while accurate, is much slower.

2. **Uric acid,** like urea, is a product of nitrogenous metabolism, and is normally excreted in the urine in small amounts (0.4 to 1.4 gramme daily). Frequently it is precipitated in the form of characteristic crystals as the result of high acidity of the urine, its poverty in mineral salts, its long standing, or because of the excessive amount of the uric acid present. The mere occurrence of such deposit does not necessarily indicate that an excess of the acid is present. Being a normal constituent of urine, chemical demonstration of its presence is of no clinical significance, and its quantitative determination is rarely demanded. Its important relation to the gouty diathesis may, however, render such an analysis desirable; the process, which is somewhat intricate, is detailed in the special works on urinalysis.

3. **Indican,** normally present in the urine is small quantities, becomes greatly increased as the result of the extensive decomposition of albuminous substances in the small intestine, *e. g.*, in cases of peritonitis or intestinal obstruction. It is increased, but in lesser degree, in connection with suppurative processes and nervous diseases. For its detection, pour 4 c.c. of hydrochloric acid into a small flask and while stirring add from 10 to 20 drops of urine. If the proportion of indican present is about normal, the resulting color will be yellow; if excessive, the acid will turn violet or blue. If no coloration appears in a minute or two, there is certainly no excess present.

4. **Xanthin, creatinin, hippuric acid,** various pigments, etc., while present in normal urine, are rarely of sufficient importance to demand chemical identification. The quantitative determination of the ethereal sulphates in their relation to the total sulphates has been recommended as a reliable index to the intensity of putrefactive changes within the intestine.

The technique of these tests will be found in works devoted to urinary analysis.

5. **Inorganic constituents** of the urine, chiefly chlorides, carbonates, sulphates, and phosphates in combination with ammonium, potassium, calcium, and magnesium, are not, as a rule, of clinical importance. The chlorides are diminished in all acute febrile diseases, gradually increasing after the crisis; the latter may, therefore, constitute a favorable prognostic sign. In pneumonia the chlorides may totally disappear from the urine, and this is often an ominous sign. Their presence and percentage is best determined by the centrifugal method. Fill the percentage tube which accompanies the centrifuge with urine to the 10 c.c. mark, add 15 drops of nitric acid, and fill to the 15 c.c. mark with silver nitrate solution (℥j to ℥j). Mix the contents by inverting the tube, revolve it at moderate speed for three minutes, and the bulk percentage of the precipitated chlorides can be read from the scale on the side of the tube. Normally it is from 10 to 12 per cent.

PATHOLOGICAL CONSTITUENTS.

Albumin may be present in the urine as a result of lesions of the renal epithelium, blood changes, and alterations in blood pressure; one or all of these conditions may be present in a single case. Nominal traces of albumin may be found in any urine by delicate testing, and a *physiological albuminuria* may follow richly albuminoid meals, intense physical strain, or a hot bath. If the urine contains blood or pus, the albumin of the plasma is dissolved out and filtration will not remove it; this constitutes a *spurious albuminuria.*

Pronounced and persistent albuminuria is, however, indicative of a renal lesion, and its discovery should be followed by systematic search for confirmatory evidence in the form of casts. The specimen of urine selected for this test should be freshly voided, preferably *not* that passed on awakening in the morning, and it must be carefully filtered.

1. *Heat Test.*—Boil the urine in a test-tube. Should cloudiness appear, which is not removed by the addition of a drop or two of acetic or nitric acid, albumin is present.

Or, add two drops of acetic acid to a test-tube full of urine and boil the upper portion. Cloudiness of the boiled portion indicates albumin.

2. *Nitric Acid Test* (Heller's).—Hold a test-tube containing a small quantity of pure nitric acid in an almost horizontal position, and allow a similar quantity of urine to trickle gently down the side of the tube and float upon the surface of the acid. An opalescent zone at the point of contact indicates albumin; if it is not at once perceptible, set the tube aside and examine it again in half an hour.

3. *Ferrocyanic Test.*—Into a test-tube pour from 15 to 30 drops of acetic acid, and add twice that quantity of potassium ferrocyanide solution (5%). Add sufficient urine to half fill the tube, close the end of the latter with the thumb and invert it three or four times in order to mix the reagents, and then stand it in a good light. If even a trace of albumin is present, a milk-like turbidity will appear within two minutes.

4. *Picric Acid Test.*—Upon the surface of some urine in a test-tube float a small quantity of a saturated watery solution of picric acid; if there is no opacity at the level of contact, albumen is certainly absent.

5. *Quantitative estimation of albumen* is best accomplished with the centrifuge. Fill the graduated tube with urine to the 10 c.c. mark, add 3 c.c. of potassium ferrocyanide solution (10%), and 2 c.c. of acetic acid, and mix them by inverting the tube several times. Revolve until all the albumin has settled; each $\frac{1}{10}$ c.c. represents 1% of albumin.

Sugar may appear in the urine as the result of cerebral or bulbar disease, etc., but its persistence in detectible quantity is the principal and sometimes the only symptom of diabetes mellitus.

1. *Reduction Tests.*—Grape sugar has the property of reducing certain substances (*e. g.*, copper oxide) to a lower state of

oxidation. Of several tests based upon this fact (Trommer's, Moore's, Fehling's) the best is:

Haines' Test.—Take pure copper sulphate, 30 grains; distilled water, ½ ounce; make a perfect solution and add pure glycerine, ½ ounce; mix thoroughly, and add 5 ounces of liquor potassæ.

Gently boil about one drachm of this solution in a test-tube, add from 6 to 8 drops of the urine, and again boil. If sugar is present, a dense yellowish precipitate appears.

2. *Fermentation Test.*—As a result of the fermentation set up by yeast, grape sugar is broken up into alcohol and carbon dioxide. This method is most reliable and should be used in all doubtful cases. By using the saccharimeter of Einhorn the test is made quantitative. Mix 10 c.c. of the urine in a test-tube with a bit of compressed yeast the size of a bean, shake well and pour the mixture into the fermentation tube. Incline the instrument until the fluid completely fills the graduated tube, and stand it in a warm room for twenty-four hours. If sugar has been present, carbonic acid will then have collected in the tube, the graduations indicating the percentage of the sugar.

This test is not reliable when more than 1 per cent. of sugar is present. On this account it is well to previously dilute the urine; a specific gravity of 1018 to 1022 necessitates double dilution, 1022 to 1028 a fivefold dilution, and 1028 to 1038 a tenfold dilution. This requires, of course, a corresponding multiplication of the result obtained by the test.

Accurate quantitative results may be obtained by titration with Fehling's or Purdy's solution, or by the use of the polariscope.

Blood may color the urine either by the admixture of its corpuscles (hematuria) or by the presence of its free coloring matter in solution (hemoglobinuria). The distinction is best made by microscopic examination.

Bile in the urine has the same significance as jaundice elsewhere. Icteric urine is dark brown in color with yellow

foam. If a few drops of such a urine and a few drops of fuming nitric acid be permitted to flow together on a white plate, at the point of contact a play of color—green, blue, violet, red, and yellow—may be observed (*Gmellin's test*).

Acetone appears in the urine abundantly after extensive albuminoid disintegration, such as occurs in high fever, severe anemia, and some cases of cancer. In severe and advanced cases its occurrence may immediately precede the more dangerous diaceturia.

Test.—Dissolve 20 grains of potassium iodide in a drachm of liquor potassæ and boil; then place the solution in a test tube and upon its surface float the suspected urine. At the point of contact phosphates are precipitated and if acetone is present yellow points of iodoform appear.

Diacetic acid is always of serious significance, and its appearance in the urine of diabetics is usually a prelude to coma and death. It may occur also at the height of acute fevers or as the sole evidence of an auto-intoxication. In either case it is of grave significance.

Test.—To some freshly-voided urine add a few drops of a ferric chloride solution. If phosphates are precipitated, filter them off, and to the filtrate add a few drops more of the ferric chloride solution. A dark red color, which disappears on boiling, indicates the presence of diacetic acid.

MICROSCOPICAL EXAMINATION OF THE URINE.

Sedimentation of the urine was accomplished formerly by setting aside the specimen in a carefully-covered conical glass for twenty-four hours. This method may still be pursued, but in general it has been abandoned for the centrifuge, by the use of which a profuse sediment can be obtained in a few minutes, and if necessary the examination can be completed before the patient leaves the consulting room.

Before sedimentating the urine ascertain its reaction, and if it is cloudy discover whether the turbidity is due to phos-

phates or urates. If the latter are present in an amount sufficient to obscure the other sediments it may be well to heat the urine moderately in a test tube, then sedimentate it rapidly, and examine the specimen on a warmed slide before cooling permits the reappearance of the urates.

In using the centrifuge fill the tube with urine to within half an inch of the top, and see that the other tube is filled to a corresponding point. Then revolve the tubes at moderate speed for two or three minutes. As a rule, this affords sufficient sediment, but if on inspection the amount of the latter appears insufficient, withdraw the upper portion of the urine without disturbing the sediment, fill the tube again with fresh urine, and rotate as before. If necessary this procedure can be repeated several times, or until enough sediment has been secured. Then carry the point of a pipette to the bottom of the tube and draw up about 5 drops of the sediment, and place it upon a clean slide. A cover-glass is unnecessary. Examine first with a low power, $\frac{2}{3}$ or $\frac{3}{4}$ objective, reserving the higher power for a more detailed inspection of the objects discovered.

CHEMICAL SEDIMENTS.

In *acid* urine the following sediments occur:

Amorphous urates (quadriurates of sodium, potassium and ammonium) appear as a reddish, granular looking sediment, the so-called "brick dust deposit." It is readily dissolved by heat, while the addition of a little hydrochloric acid will cause uric acid to crystallize out. The presence of amorphous urates indicates only acidity and concentration of the urine.

Uric acid appears as crystals yellow or brown in color, in consequence of the urinary pigment absorbed by them, and varying in shape from rhombic prisms to cylinders, rosettes, etc. It becomes re-dissolved on the addition of potassium hydrate, but not on heating, and re-crystallizes out on the addition of hydrochloric acid.

Uric acid sediments are often the result of febrile states accompanied by concentrated and therefore more acid urine. Their persistence, however, suggests an actual excess of uric acid; and on account of the importance of the latter in its re-

FIG. 16—Uric Acid Crystals. (Salinger-Kalteyer.)

lation to the uric acid diathesis, repeated quantitative analyses may be desirable.

Calcium oxalate appears in the urine as colorless octahedral crystals or minute dumb-bells. A small number of these may

FIG. 17—Various Forms of Calcium Oxalate Crystals. (Salinger-Kalteyer.)

be normal; an excessive quantity possesses a significance allied to that of uric acid (oxalic acid diathesis).

Cystin rarely appears in the urine. When present, it as-

sumes the form of hexagonal crystals which are readily soluble in ammonia.

Leucin is sometimes seen in the form of yellowish refractive spheres which resemble oil globules, but unlike the latter are insoluble in ether. **Tyrosin** crystallizes in colorless sheaves of fine needles. These two sediments appear in the urine, usually together, as a result of phosphorus poisoning or acute yellow atrophy of the liver.

In *alkaline* urine the following sediments occur:

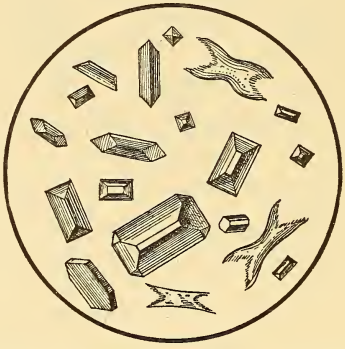

FIG. 18—Crystals of Triple Phosphate. (Salinger-Kalteyer.)

Ammonio-magnesium ("triple") **phosphate,** colorless coffin-shaped crystals readily soluble in acetic acid.

Calcium phosphate, either "amorphous," *i. e.*, small colorless granules deposited as a white sediment in alkaline urine, or rarely "stellar phosphate," crystalline rods arranged in stellar or rosette form.

Ammonium urate, dark-brown spherical masses studded with fine spicula—"thorn-apple crystals."

These alkaline deposits *indicate nothing but the decomposed state of the specimen* of urine.

ANATOMICAL SEDIMENTS.

Blood corpuscles may appear in the urine; they may exhibit the characteristic reversal of light and shade at their margins while the lens is being focussed up and down, but they are pale and do not form rouleaux. *Hematuria* is always pathological, indicating hemorrhage from the kidney, ureter, bladder, or urethra; and among the possible causes of the latter may be included congestion, inflammation, cancer, tubercle, syphilis, stone, and traumatism. As a rule, the brighter the color of the blood, the nearer its source to the meatus; and the more intimate its admixture with the urine and the darker its color, the higher in the urinary tract is its source. Associated symptoms and physical signs will aid in localizing the lesion. *Blood pigment* may also appear in the urine (*hemoglobinuria*) turning it dark or smoky in color. It is the result of corpuscular destruction within the blood vessels, the pigment being set free and eliminated by the kidneys; the causes are usually toxic (poisoning by carbolic acid, potassium chlorate, etc., or infectious fevers, such as malaria, yellow fever, etc.), but it may occur as a specific disease *(paroxysmal hemoglobinuria)*. It is distinguished from hematuria by the failure to discover any corpuscles on microscopical examination, while the *guaiac test*, which consists in overlaying the urine with equal parts of tincture of guaiac and hydrogen dioxide, shows by an indigo-blue ring above the white deposit that blood-pigment is present.

Pus corpuscles (leucocytes) may appear sparsely in normal urine, but if abundant *(pyuria)* they indicate suppuration in, or rupture of an abscess into, some point in the urinary tract. In women, however, the pus may be due to leucorrhea, in which case it is associated with flat epithelial cells from the vagina. Otherwise, purulent urine, if acid, generally comes from the kidneys; if alkaline, from the bladder. The pus corpuscles appear as circular, granular discs, with one or more nuclei. In the fresh state they exhibit ameboid movement, but in the urinary sediment they are usually dead.

Epithelium is found in small amount in every specimen of healthy urine, but as a result of diseased states it may be thrown off in much larger quantity. Formerly it was believed that the special form of epithelial cell found in the urine was a valuable aid in locating the lesion, but this is only partly true. Three varieties of cells may be recognized, *viz.:*

Round cells, derived from the uriniferous tubules and the deep layers of mucous membrane in the renal pelvis, bladder and male urethra.

Columnar cells, irregular and elongated, mostly derived from the superficial layer of mucous membrane in the renal pelvis.

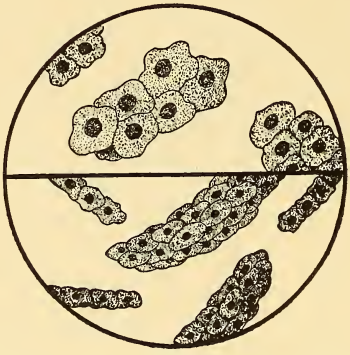

FIG. 19—Squamous Epithelium, as it Appears in the Urine. (Upper Half.) Epithelial Casts. (Lower Half.) (Salinger-Kalteyer.)

Flat cells, large and somewhat rounded, derived chiefly from the bladder and vagina.

Casts are probably due to the exudation into the renal tubules of the coagulable portion of the blood in which detached and altered anatomical elements become entangled. They are washed from their tubular mold by the urine, in which they appear as :

Hyaline casts, homogeneous, transparent structures, often with ill-defined contours. Their presence is not positive proof of nephritis; they are present in association with other casts in all forms of renal inflammation, and occur alone in cases of

contracted kidney, passive hyperemia of the kidney, and fevers. A larger form, yellowish in appearance and stained brown by the addition of iodine, is the so-called "waxy cast" which was formerly supposed to indicate amyloid degeneration

FIG. 20—Hyaline Casts. (Salinger-Kalteyer.)

of the kidney, but is now known to occur in all forms of chronic nephritis.

Epithelial casts, consisting of epithelial cells agglutinated into a cylinder, are the result of nephritis involving the tubules.

FIG. 21—Coarse and Fine Granular Casts. (Salinger-Kalteyer.)

Granular casts result from the metamorphosis of epithelium or blood cells; they may be light or dark and coarsely or finely granular. Their presence indicates a chronic degenerative lesion of the kidney.

Blood casts, composed of agglutinated corpuscles, indicate hemorrhage into the tubules.

Pus casts occur rarely in connection with suppurative renal disease.

Many casts are of a composite type, granular matter, epithelial cells or blood being embedded in a hyaline matrix. Cylindroids, long, ribbon-like mucous threads of uncertain significance, must not be mistaken for casts; and epithelial cells, corpuscles, urates, etc., may be grouped into false casts which can be distinguished only by experience.

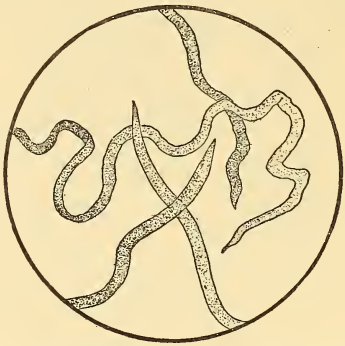

FIG. 22—Cylindroids. (Salinger-Kalteyer.)

UREMIA.

Etiology.—The condition is one of toxemia due to the retention of certain waste matters normally excreted by the kidneys.

Symptoms may appear abruptly (*acute uremia*) or develop gradually (*chronic uremia*). They may be limited to one or two phenomena, such as headache and dyspepsia, or may include the more pronounced forms.

Cerebral Symptoms. — Headache, persistent drowsiness, vertigo, delirium, coma, convulsions, sudden blindness (often without retinal change), and transient paralyses.

Respiratory Symptoms. — Dyspnea ("uremic asthma"), Cheyne-Stokes respiration.

Abdominal Symptoms.—Dyspepsia, pain, nausea, vomiting hiccough, diarrhea, or constipation.

General Symptoms.—Cramps, particularly in the calf-muscles at night; itching of the skin.

Diagnosis.—A knowledge of the renal condition will lead to instant recognition of the cause of these symptoms. In doubtful cases, the patient being comatose or in convulsions, it may be noted that the breath has the odor of urine, the arterial tension is high, the urine is suppressed, and the small amount obtainable from the bladder by catheterization contains albumin and casts.

Prognosis.—Grave. From the uremia of acute nephritis permanent recovery may occur; in cases of chronic nephritis recovery, while possible, can be but temporary.

Treatment.—Stimulate the organs of elimination; the skin, by the use of warm baths or heat (hot air, hot bottles); the bowels, by cathartics (croton oil, two drops in a drachm of olive oil or glycerine; elaterium, gr. ¼ in solution); and the kidneys themselves, by the administration of *cuprum ars.*, five grains of the second decimal trituration, repeated in one hour if necessary, or continued in grain doses every two hours if the symptoms are not acute. *Mercurius corrosivus* may be similarly useful at times.

ACUTE HYPEREMIA OF THE KIDNEY.

Etiology.—Renal congestion may be due to:

1. Poisons (cantharides, turpentine, arsenic, etc.).
2. Infections (common to most fevers).
3. Compensation (after removal of one kidney).
4. The first stage of nephritis.

Symptoms.—Scanty urine of high specific gravity and darkened color generally constitutes the only evidence of renal congestion. Unless inflammatory changes (acute nephritis) supervene, no direct treatment is necessary.

CHRONIC HYPEREMIA OF THE KIDNEY.

Etiology.—Passive congestion results from venous obstruction, which may be:

1. Systemic (chronic disease of heart, lungs, or liver).

2. Local (obstruction to renal veins by thrombus or pressure of tumor).

Pathology.—The kidney becomes enlarged, firm, smooth, and bluish-red (cyanotic induration), the veins are engorged with blood, and nephritis may result finally.

Symptoms.—The urine is scanty, of high specific gravity, and contains some albumin and a few narrow hyaline casts. The general symptoms are those of the primary disease.

Diagnosis.—Passive congestion of the kidney accompanies all uncompensated valvular heart lesions. It must be distinguished from chronic interstitial nephritis, *q. v.*

Treatment must be directed to the primary lesion.

THE CLASSIFICATION OF BRIGHT'S DISEASE.

- Nephritis.
  - Acute.
    - Parenchymatous. — Recovery usual; death not infrequent; may terminate in chronic parenchymatous form.
    - Interstitial suppurative or pyelo-nephritis. — Acute surgical kidney; rare; due to ascending infection.
  - Chronic.
    - Parenchymatous.
      1. "Large white kidney," which may become
      2. "Small white kidney" or may be associated with the
      3. Amyloid kidney.
    - Interstitial suppurative or pyelo-nephritis (chronic surgical kidney.)
    - Imterstitial (non-suppurative.)
      1. Primary.
      2. In association with general arteriosclerosis.

ACUTE PARENCHYMATOUS NEPHRITIS.

(Acute Diffuse, Desquamative, Tubal or Catarrhal Nephritis.)

Etiology.—Acute inflammation of the renal Parenchyma results from the passage through the kidney of irritants, including—

1. Bacterial toxins, in connection with an acute infectious disease (scarlatina, etc.).

2. Bacteria.

3. Chemical poisons (cantharides, turpentine, and other substances eliminated by the kidneys).

4. Exposure to cold and damp, probably through its effect on metabolism, may indirectly induce nephritis.

Pathology.—The kidney becomes swollen and bright red in color at first; later it is paler and and mottled. The capsule is non-adherent. The inflammatory process consists of—

1. *Hyperemia*, intense in character, which causes enlargement of the kidney even to the point of rupturing its capsule; and by pressure occludes the tubules, prevents excretion, and may rupture the blood vessels, with resulting hematuria.

2. *Exudation* into the tubules, occluding their lumen. The desquamated epithelium together with blood corpuscles becomes enmeshed in this exudate and the whole appears in the urine as a cast.

3. *Hyperplasia* of the connective tissue begins at the end of 30–40 days.

Symptoms.—1. *Urinary changes.*

a. Reduction in the quantity, even suppression.

b. Presence of albumin in large quantities.

c. Presence of blood. A large quantity causes a dark, smoky appearance of the urine; but a small amount requires the microscope for its detection.

d. Presence of casts (epithial, granular, blood, and hyaline).

e. Reduced solids. The decrease in the quantity of urine renders it concentrated and of high specific gravity (1020–1040), but the total daily elimination of solids is much lessened.

2. Rapid and progressive *anemia*.

3. *Dropsy*. This usually begins about the face, but general anasarca may ensue.

4. *Gastro-intestinal disturbances*. Nausea and vomiting are quite common.

5. *Uremia* may develop at any time.

Duration.—Acute nephritis tends to recover in from one to six weeks; but it may merge into a sub-acute or chronic form.

Diagnosis.—The urinary examination is conclusive. Acute nephritis may occur in the course of chronic nephritis, but the cardio-vascular changes will indicate the pre-existence of the latter. The simple albuminuria of fever shows neither blood nor epithelial casts in the urine.

Prognosis.—Guardedly favorable. Uremic symptoms and suppression of urine are unfavorable signs.

Treatment.—1. Absolute rest in bed, with liquid diet.

2. Reduction of the renal congestion may be favored by stimulating the skin (hot baths, 105°–120°, for ten or twenty minutes once or twice daily; or dry cups may be applied to the back); and by relaxing the bowels (Hunyadi water). Pure water in large quantities should be taken by the patient.

3. Medicines. *Aconite* is generally indicated in the early stages, while renal congestion is intense; occasionally *veratrum viride* may be preferable. In children symptoms of cerebral irritation, such as convulsions, with dry skin, etc., may indicate *belladonna*.

Later, in the exudative stage, *rhus toxicodendron* is often clearly indicated and may be the only remedy required. Should the degree of dropsy be considerable, however, preference should be given to *cantharis;* while cases with

little dropsy but much blood in the urine are benefited by *turpentine*.

In more advanced cases, general anasarca requires the administration of *mercurius corros.*, *apis*, or *arsenic*. It may be necessary to remove the dropsical accumulation by mechanical means (capillary drainage) or by the administration of diuretics (infusion of digitalis or of apocynum, 20–30 minims every three hours). Anemia may justify the use of iron preparations.

CHRONIC PARENCHYMATOUS NEPHRITIS.

(Chronic Diffuse, Tubal, or Catarrhal Nephritis.)

Etiology.—A chronic inflammatory process particulary affecting the renal parenchyma may be :

1. A sequel of acute nephritis.

2. A result of the same cause long continued, *i. e.*, passage of some irritant (bacterial, toxins, alcohol, uric acid, etc.).

3. A complication of pregnancy.

Pathology.—The disease is primarily an inflammation of the tubular epithelium, with a secondary interstitial fibrosis. Two stages are distinguishable, viz.,

1. The *large white kidney*. The tubular epithelium is swollen and degenerated, the lumen of the tubules is more or less occluded with debris, and bands of interlobular connective tissue form; as a result the arteries are compressed and the cortex is thickened and anemic, while the pyramids are dark in color.

2. *The small white kidney*. Later, contraction of the connective tissue renders the organ smaller and lobulated. The tubes are choked as before, and their constriction occasions minute retention-cysts.

In the first stage the capsule strips off easily, though sometimes it drags a little of the parenchyma with it; in the second stage it is largely adherent. Amyloid degeneration often supervenes upon this form of nephritis.

Symptoms.—While similar to the acute form, this variety is much more insidious in its development.

1. *Anemia* is more marked than in the acute form, is of a progressive character, and is associated with weakness and emaciation.

2. *Dropsy* is also prominent, appearing first about the eyes, and then becoming general.

3. *Gastro-intestinal derangements* are common.

4. *Uremia* is less frequent than in the acute form.

5. Thoracic *complications* often occur; they include bronchitis, pneumonia, pulmonary edema, pleurisy, and peri- and endocarditis.

6. *Urinary changes.*

a. Quantity and specific gravity about normal, but varying.

b. Color pale; whitish sediment common.

c. Albumin present in large amount (1–5%).

d. Urea diminished.

e. Casts: broad; finely granular, fatty and hyaline.

f. Blood corpuscles, if present, suggest an intercurrent acute nephritis.

Should contraction occur, the urine approximates in character that of chronic interstitial nephritis.

Diagnosis is usually easy, the urinary changes, together with the marked dropsy and anemia, distinguishing this from the interstitial form. The amyloid variety can be recognized only in connection with a history of suppuration and the presence of amyloid changes in other organs.

Prognosis.—Generally unfavorable, death occurring within two years. Careful treatment may, however, partially arrest the disease; and should contraction occur there may appear to be a practical restoration to health.

Treatment.—1. Until the symptoms abate, treat as an acute case. Then allow a mixed diet, limiting it again if active symptoms or gastro-intestinal irritation appear.

2. Hot baths given daily at bedtime are of great benefit.

3. Keep the bowels relaxed.

4. Administer pure, preferably distilled, water freely.

5. Avoid chill; let the patient wear woolen underclothing and if necessary remove him to a warm, moist climate in winter.

6. Uremia, dropsy, and anemia should be treated symptomatically as in the acute form.

7. Medicines. *Cantharis* is frequently indicated by scanty, highly albuminous urine associated with dropsy, even though other symptoms be lacking. *Mercurius corros.* has less effect upon dropsy, but is well suited to cases in which dropsy and other acute symptoms have subsided but the urine remains albuminous and rather scanty. Anemia, gastro-intestinal irritation, and a history of syphilis further indicate it. The latter may also suggest the use of *kali iod.* or *aurum.* Dropsy with a tendency to uremic symptoms calls for the use of *cuprum ars.*

CHRONIC INTERSTITIAL NEPHRITIS.

(Red Granular, Contracted, or Gouty Kidney.)

Etiology.—The causes, while often obscure, include:

1. Gout. The uric acid diathesis may be associated with alcoholism.

2. Lead. Chronic plumbism induces gouty changes.

3. Prolonged passive congestion, the result of heart disease.

Heredity, infectious diseases (scarlatina, malaria, rheumatism, syphilis) and digestive disorders have some causative influence. It occurs in middle life, and is usually associated with sclerosis of other organs; and it is a constant feature of old age.

Pathology.—The disease is essentially a connective tissue hyperplasia which destroys the tubules by compression. The

kidney is small, red, and granular, with a greatly atrophied cortex and an adherent capsule. Microscopically, great bands of intertubular connective tissue are seen; by this contraction the tubules are in part obliterated, leaving dilated portions (cysts). General arterio-sclerosis, with hypertrophy of the heart, is invariably associated.

Symptoms.—The disease is extremely insidious; there may be:

1. Slight gradual *loss of strength*, with digestive disturbances.

2. *Cardio-vascular changes:*

   a. Rigid arteries with a high tension pulse.

   b. Hypertrophy of the left ventricle, with a sharp accentuation of the second sound at the aortic orifice.

3. Slight *edema*, under the eyes or about the ankles.

4. *Urinary changes*. The urine is increased in quantity, pale, of low specific gravity (1003–1015), and the excretion of urea is diminished. There may or may not be a small amount of albumin; and casts, mostly hyaline, are usually present.

The patient commonly has to arise at night to micturate; and should this or some other symptom lead to examination of the urine, the disease is easily recognizable. Many cases, however, remain unsuspected until the onset of:

5. *Uremia* (headache, vertigo, drowsiness, coma, convulsions, etc.).

6. *Dimness of vision* (albuminuric retinitis).

7. *Hemorrhage*, cerebral or elsewhere.

8. *Dilatation of the heart*, with dropsy, a diminished quantity of urine, dyspnea, and reduplication of the first sound, may occur in the later stages.

Diagnosis.—Examination of the urine usually insures discovery of the disease.

Passive renal hyperemia, the result of uncompensated heart disease, may be distinguished by the history of the primary

disease, the less marked arterio-sclerosis, and the fact that urea is little diminished and the urine contains only a few small hyaline casts; the condition usually disappears upon the restoration of cardiac compensation.

Chronic parenchymatous nephritis is accompanied by marked dropsy, occurs earlier in life, presents little cardio-vascular change, and the urine is highly albuminous with broad granular and hyaline casts.

Prognosis.—While the damage is irremediable, the process may be checked; and if sufficient renal parenchyma remains intact, life may be prolonged indefinitely. Persistent diminutition of urea (below 175 grains per day) or actual uremic symptoms render the outlook unfavorable.

Treatment.—1. Arrest the change in the blood vessels. This is possible if gout be the cause, the treatment being dietetic (restriction as to nitrogenous foods, etc.), and hygienic (maintenance of body warmth by clothing or climate; fresh air, moderate exercise and avoidance of excitement).

2. Control symptoms as they arise. Such complications as bronchitis, pneumonia and pleurisy must be treated symptomatically; and the management of uremia has been considered elsewhere.

3. Medicines. *Chloride of gold*, 2x in a fresh watery solution, is of great value in the early stages, especially when various neurotic symptoms are present. *Rhus tox.* possesses a decided eliminative value, and is frequently indicated by gouty symptoms. *Plumbum*, which offers a perfect simile to the conditions present, is frequently suggested by digestive and abdominal symptoms. *Mercurius corr.* is suited to intercurrent acute or subacute exacerbations, and to syphilitic cases. *Glonoin* is invaluable when the high arterial tension occasions throbbing headache, etc. *Phosphoric acid* (general neurasthenic symptoms with excessive urination), *nux vomica* (digestive disorders) and *arsenic* (thirst, debility, and urinary symptoms) are frequently useful.

AMYLOID KIDNEY.

Etiology.—Lardaceous degeneration of the kidney is a sequence of prolonged suppuration or cachexia (bone disease, tuberculosis, syphilis).

Pathology.—Waxy infiltration begins in the capillaries of the malpighian tufts, involves blood vessels and tubules, and is associated with diffuse nephritis. The kidney becomes large, smooth, anemic, and "bacon-like" on section, the degenerated areas staining a mahogany-brown on the application of an iodine solution.

Symptoms.—The general symptoms of chronic parenchymatous nephritis are added to those of the causative disease, and in addition amyloid changes may be detected in the liver, spleen and often the intestines. Dropsy is moderate. The urine is pale, copious, of low specific gravity (1005–1015) and highly albuminous, with a moderate number of hyaline or waxy casts.

Diagnosis.—The history and the discovery of simultaneous enlargement of the liver and spleen are necessary for positive diagnosis. Parenchymatous nephritis lacks the causative factors and presents more general dropsy; but the two forms are often associated. Interstitial nephritis has little dropsy, slight albuminuria, and scanty urinary sediment.

Prognosis.—Recovery is impossible, but occasionally control of the causative may check the renal degeneration.

Treatment must be directed toward the primary disease, while the kidney lesion should receive the attention recommended for the other forms of nephritis.

PYELONEPHRITIS.

("Surgical Kidney;" Suppurative Interstitial, Obstructive, or Ascending Nephritis.)

Etiology. — The kidney may become infected by pyogenic organisms through:

1. The genito-urinary tract, as the result of the inflammation attendant upon the passage of calculi or even uric acid "sand," as the sequence of unclean surgical interference, or by extension upward of a ureteritis, cystitis, or urethritis.

2. The blood-vessels, *e. g.*, by septic emboli, in connection with pyemia, etc.

3. Renal affections, such as tuberculosis, carcinoma, etc.

4. Toxic irritants (cantharis, copaiba, and other irritants given as diuretics, etc.).

5. Traumatism (penetrating wounds involving the kidney) and extension of neighboring abscesses.

Pathology.—The suppuration, usually advancing from below, first involves the renal pelvis (*pyelitis*) and then the substance of the kidney itself. Foci of suppuration may develop in one or both kidneys, forming multiple abscesses which later unite and finally burst into the pelvis of the kidney, whence the pus is discharged with the urine. In advanced cases the kidney may be simply a collection of purulent sacs or one large abscess. Distention of the renal pelvis and calyces with pus (*pyonephrosis*) may result from partial obstruction of the ureters.

Symptoms—The early symptoms are indefinite. When the suppurative process remains confined to the renal pelvis (*pyelitis*), the clinical manifestions may be limited to slight fever, some tenderness in the region of the kidney, and small amounts of pus and mucus in the acid, slightly albuminous urine. With extensive involvement of the renal parenchyma, however, there is apt to be:

1. *Aching* or *pain* in the region of the kidney.

2. *Rigors*, *sweats*, and a *septic temperature* range.

3. Rapid *emaciation*, gastric irritability, and a typhoid state.

4. The *urine* becomes diminished in quantity, of low specific gravity, and albuminous. It contains pus, blood, bacteria, and perhaps casts. The amount of albumin is in direct proportion to the quantity of pus and blood present. The excretion of urea is diminished.

Diagnosis.—In many cases the renal lesion is masked by the symptoms of the primary disease (cystitis, nephrolithiasis, etc.). Repeated examinations and urinalyses may be necessary before a diagnosis is possible; and the latter usually rests upon the discovery, in connection with possible causes and vague symptoms, of pus, blood, and perhaps caudate epithelial cells from the pelvis of the kidney in the urine.

Prognosis.—In all but the mildest cases of pyelitis, the prognosis is extremely unfavorable. Life may be prolonged, however, and if the cause (*e. g.*, obstruction of the ureter) is removable, recovery is possible.

Treatment.—The physician should endeavor, by prompt attention to urethral and cystic inflammations, and by cleanliness in all instrumentation about the genito-urinary tract, to guard against the possibility of renal infection. Upon the appearance of any symptoms suggesting the existence of pyelitis or pyelonephritis, the patient should be placed at rest, hot fomentations applied, and his diet restricted to milk and other bland liquids. A medicine having a local antiseptic action on the kidney should be given: *cystogen*, or *urotropin*, gr. v. t. i. d., *saccharin*, gr. ij. t. i. d., or *methylene blue*, gr. j. in caps. t. i. d. The suppurative process affords indications for such remedies as *mercur. corr.* or *hepar;* the renal changes may be met by the administration of *terebinth*, *cuprum arsen.*, or *cantharis;* and general septic conditions sometimes afford indications for *arsenic*, *carbolic acid*, *baptisia*, or *lachesis*.

TUBERCULOSIS OF THE KIDNEY.

Etiology.—Renal tuberculosis may develop—

1. In connection with acute general tuberculosis.

2. In connection with tuberculosis elsewhere in the genito-urinary tract. Tuberculosis of the epididymis, seminal vesicles, prostate, or bladder usually pre-exists. Often it affects adolescent males after gonorrhea, especially if the latter has been complicated with epididymitis; and in these cases it involves the ureter first, thence ascending to the pelvis and finally involving the parenchyma of the kidney.

3. Primary renal tuberculosis is rare. Usually it is unilateral.

Pathology.—1. In connection with general tuberculosis, miliary tubercles may develop in the kidney, but they occasion no special symptoms.

2. Tuberculosis ascending from the lower portions of the tract is apt to be double. It occasions pyonephrosis or renal or peri-renal abscess, frequently followed by septicemia.

3. Primary renal tuberculosis occurs as an infiltration of the parenchyma; the areas undergo caseation and softening, the organ becoming honey-combed with cavities, and the pus is discharged through the ureter.

Symptoms.—The symptoms are vague at first; there may be pus or blood in the urine, evening pyrexia, some tenderness or pain in the loin, and in general the signs of pyelonephritis. The **diagnosis** rests upon:

1. The discovery of tuberculosis elsewhere.

2. The absence of any cause for pyelonephritis.

3. The discovery of tubercle bacilli in the urine.

Prognosis.—Unfavorable.

Treatment.—An early diagnosis, which can be assured only by the repeated examination of every pus-containing urine for the tubercle bacillus, should be followed by an immediate nephrotomy.

NEPHROLITHIASIS.

(Renal Calculus; Stone in the Kidney.)

Etiology.—The urinary solids are precipitated about a nucleus, often of epithelial cells, mucus, or blood clot, to form a concretion.

Varieties.—1. Uric acid (smooth, firm, brown, or black; often multiple).

2. Oxalate of lime (uneven, hard, and brown; "mulberry calculus").

3. Phosphates (large, soft, and white).

4. Cystin and xanthin (rare).

Course.—1. The stone may remain quiescent in the kidney tubules or substance, growing to a considerable size and occasioning vague symptoms until it has bored out for itself a pocket—a so-called "nesting stone." It may enter the pelvis, where it grows and often assumes the shape of that cavity.

2. The stone may pass down the ureter, with or without renal colic.

3. The stone may become impacted in the ureter, blocking the out-flow of urine and causing hydro- or pyonephrosis, suppression, or abscess.

4. The stone may break into the retroperitoneum or the peritoneal cavity from either the kidney or the ureter.

Symptoms.—The symptoms vary according to the situation and course of the stone.

1. The existence of a *stone in the kidney* causes few symptoms unless it is free in the pelvis or entering the ureter. Under these circumstances it causes:

(*a*) *Pain*, which may be *direct*, varying from mere uneasiness to severe paroxysmal or continuous pain in the region of the kidney, associated with tenderness and rigidity, and *aggravated by motion;* or *reflex*, extending to the groin,

testicle, and inner side of the thigh, along the course of the genito-crural nerve, or along the line of the ureter.

(*b*) *Hematuria*, irregular in its occurrence but always *increased by exercise*. The blood may appear clear or smoky in the urine, or it may occur in the form of worm-like clots.

(*c*) *Cystic symptoms* may alone be present, micturition being frequent and the urine containing pus and crystals.

(*d*) *Digestive disturbances*, vague in character, may accompany the renal disease.

2. *Passage of the stone* down the ureter is usually accompanied by *renal colic*. At the moment when the stone enters the ureter the patient is suddenly seized with agonizing pain in the loin which radiates down the line of the ureter into the root of the penis, the prostate, the scrotum, or the thigh. The terrific pain may compel the patient to writhe, rolling on the floor, and bending double in order to relax the ileo-psoas and lumbar muscles; and symptoms of shock and even collapse, with cold sweat, sometimes ensue. Vesical irritation, tenesmus, constipation, and vomiting frequently accompany the paroxysm. At times the symptoms strongly suggest intestinal obstruction. After a period varying from minutes to hours the pain ceases as suddenly as it began, owing to the passage or recession of the stone. Subsequently the urine may contain pus or blood.

3. *Impaction of the stone* in the ureter leads to recurrences or persistence of the colic, and, as a rule, distinct localized pain and tenderness may be located near the entrance to the bladder by rectal or vaginal palpation. The ureteral orifice is exquisitely tender, and if obstruction is incomplete, blood, pus, and crystals appear in the urine.

Diagnosis.—Stone in the kidney must be distinguished from *renal tuberculosis;* in the latter the patient's physique and family history, the usual absence of renal colic, the lack of benefit from rest or aggravation from motion, and the appearance of little blood and no crystals in the urine will generally

suffice for diagnosis. Discovery of the tubercle bacillus in the urinary sediment is conclusive.

Biliary colic is accompanied by pain radiating from the right hypochondrium into the umbilicus, into the right shoulder-tip and the angle of the scapula, with tenderness over the gall bladder or the Mayo-Robson point. Jaundice is common, and urinary symptoms and signs are absent.

Prognosis.—Because of the possible complications the prognosis must be guarded. Renal colic is rarely fatal in itself.

Treatment.—Attacks of renal colic may be palliated by the application of hot fomentations, or better, a long-continued hot general bath. Frequently the hypodermatic use of morphia, gr. $\frac{1}{4}$, with atropia, gr. $\frac{1}{120}$, is necessary. *Belladonna*, *berberis*, and *nux vomica* are medicines frequently indicated.

In the interval between attacks, the diathetic state requires careful attention. In a majority of cases the patient is gouty, and the therapeutic measures must be directed to that condition. Meats, eggs, alcoholics, etc., should be forbidden, and pure or alkaline water prescribed in large quantities. If, however, there is phosphaturia and alkaline urine, forbid an excess of vegetables and milk, allow meats, and administer dilute hydrochloric acid to acidulate the urine. The more important remedies are *calcarea*, *lycopodium*, *sarsaparilla*, and *sulphur*.

In painful and persistent cases it is advisable to explore the kidney with nephrolithotomy, evacuation of abscesses, sounding or flushing the ureter, etc. The kidney can be opened from end to end, explored, and drained or sutured with impunity. (Van Lennep.)

HYDRONEPHROSIS.

Etiology.—The renal pelvis becomes distended with retained watery secretion as the result of gradually developed obstruction to the outflow, sudden complete obstruction leading

simply to absolute suppression of urine. This gradual obstruction may be due to—

1. Impaction of a calculus in the ureter.

2. Stricture of the ureter (congenital or inflammatory).

3. Twisting of the ureter (floating kidney).

4. Pressure upon the ureter by inflammations, tumors, cicatrices, etc., from without.

5. Obstruction in or below the bladder (tumors, enlarged prostate, urethral stricture), which tends to cause bilateral hydronephrosis.

Pathology.—The pressure of the retained fluid causes—

1. Distention of the pelvis of the kidney.

2. Pressure-atrophy of the kidney tissue, which finally leaves nothing but a thin-walled cyst. Infection renders the condition one of *pyonephrosis*.

Symptoms.—1. A fluctuating *tumor* develops in the region of the kidney and enlarges downward and forward toward the groin, following the line of the ureter.

2. An excessive flow of urine, followed by a notable diminution in the size of the tumor, may be observed occasionally (*intermittent hydronephrosis*). This symptom is inconstant, however; as a rule, it occurs in those cases due to twisting of the ureter by a movable kidney.

3. No subjective symptoms are occasioned by slight distention, but as the tumor increases, *pressure symptoms*, such as a tense feeling in the abdomen, nausea, vomiting, and constipation, may appear.

4. The urine is normal in quantity, except in the rare cases of double hydronephrosis, and is free from albumin. Double hydronephrosis quickly leads to anuria, uremia, and death.

Diagnosis.—This rests upon discovery of the tumor in the flank, its enlargement in the characteristic direction, the exclusion of other abdominal tumors, and in doubtful cases examination of the aspirated fluid, which resembles weak urine and contains urea and uric acid.

Treatment.—When the obstruction is due to a stone, the loin may be massaged and the bladder distended in an attempt to overcome the block. Aspiration may be made repeatedly, but when it fails to afford permanent relief it is wise to incise and drain the sac, and to remove or overcome the ureteral obstruction by operative means if possible, or to extirpate the kidney if the latter is extensively diseased.

FLOATING OR MOVABLE KIDNEY.

Etiology.—As a result of relaxation of its surrounding tissues, one kidney, usually the right, becomes displaced and may be found as a movable tumor in any part of the abdomen. Predisposition is found in:

1. Women, six times as often as men, because of—
   a. Relaxation of the abdominal walls.
   b. Relaxation of the peritoneum.
   c. Repeated pregnancies.
   d. Tight lacing.
2. Middle life. Rarely it may be congenital.
3. Muscular exertion, such as heavy lifting.

Mobility of the kidney is frequently associated with a downward displacement of all the abdominal viscera (*splanchnoptosis*).

Symptoms.—1. Subjective sensations may be slight or wanting; often they are limited to vague "dragging" pain.

2. Reflex nervous symptoms are sometimes marked; they consist of flatulence, palpitation, neuralgic pain in thorax or abdomen, irritable bladder, dysmenorrhea, and the train of symptoms commonly attributed to uterine disease.

3. Twisting of the ureter, involving vessels and nerves, may occasion the agonizing pain and other symptoms of renal colic, and may lead to hydronephrosis.

Diagnosis.—Bimanual palpation, with the patient in various

positions if necessary, reveals a smooth reniform tumor, which is freely movable; and pressure develops a peculiar sickening pain.

A *distended gall-bladder* forms a fluctuating pyriform cyst, non-separable from the liver, which is totally unlike the firm kidney. On pressure a movable kidney frequently becomes lost in the abdomen, while the gall-bladder springs back to its previous position when that pressure is removed. Other *abdominal tumors* are more painful, have not the shape or peculiar tenderness of the kidney, are apt to have an uneven surface, and if malignant in nature they are apt to be associated with cachexia.

Treatment.—Many cases require no treatment. If the symptoms become annoying, rest on the back followed by application of a binder and pad or truss may suffice to retain the kidney in place. Should this prove ineffective, the kidney may be stitched in its normal position (nephrorrhaphy), or better, a plastic union may be secured (nephropexy).

CYSTITIS.

Etiology.—Inflammation of the bladder results from local microbic infection, due to:

1. Irritants within the bladder (calculi, foreign bodies, etc.).
2. Obstruction to micturition (stricture, prostatic hypertrophy) with resulting decomposition of the retained urine.
3. Extension of inflammation from without (gonorrhœa, etc.).
4. Toxic substances (cantharides, capsicum, etc.).
5. Traumatism (direct injury to the bladder, etc.).

Pathology. — The mucous membrane is inflamed, perhaps ulcerated or denuded, and in chronic cases it may be thickened; mucus, pus, and epithelium are discharged in large quantities.

Symptoms.—The disease may be acute (febrile) or chronic; it is accompanied by--

1. Frequent and painful micturition.

2. Tenderness or pain over the bladder.

3. Urine which contains pus and mucus (hence it is glairy), together with epithelial cells and sometimes blood.

Diagnosis.—Pyuria with vesical pain and tenesmus is pathognomonic of cystitis. *Chronic interstitial nephritis*, possibly coexisting, is indicated by the presence of casts in the urine and by the associated signs of cardio-vascular sclerosis. *Pyelitis* or *pyelo nephritis* can often be excluded with difficulty or not at all.

Prognosis.—In acute cases the prognosis is usually good; in chronic cases the outlook is less favorable, but recovery is possible if the cause can be removed.

Treatment.—The patient should be placed at absolute rest in bed, pure water should be administered freely, and a plain, preferably milk, diet must be given until active symptoms subside. The cause should be sought out and treated. *Boric acid*, five grains every four hours, or *cystogen*, gr. v t. i. d., should be administered in order to render the urine aseptic.

Aconite and *gelsemium* are indicated early in acute cases. Later, and in chronic cases, *belladonna*, *cantharis*, and *mercurius corrosivus* are often useful. *Terebinth* is a remedy of great value, particularly in cases attended with intense irritability of the bladder.

Cases dependent on obstruction demand catheterization, at times antiseptic irrigation; and occasionally incision and drainage becomes necessary.

INCONTINENCE OF URINE.

Etiology.—Incontinence of urine (enuresis) may be—

1. *Paralytic* (spinal lesions involving sphincteric centre).

2. *Irritative* (calculus, cystitis, parasites, etc.).

3. *Mechanical* (retention with over-distention; pressure from without, as of the fetal head during labor).

4. *Spasmodic* (overaction of compressor muscle, with diminished vesical capacity).

Treatment varies according to the cause. The nocturnal enuresis of children may be due to any of these conditions, and it is particularly necessary to exclude epilepsy and local irritations due to ascarides, elongated prepuce, or masturbation, before deciding the symptom to be purely neurotic. In a case of the latter class the bowels should be kept open, the evening meal must be light, and the bladder must be emptied at bedtime. This, with the administration of such remedies as *belladonna*, *equisetum*, or *sulphur*, will usually affect a cure.

Retention of urine may be neurotic (hysteria) or mechanical (obstructive) in origin. The latter may be due to stricture, enlarged prostate, impacted calculus, or pressure upon the urethra by tumor or gravid uterus. It is sometimes the only symptom of commencing peritonitis in women.

Suppression of urine is due to secretory failure of the kidney and the resulting symptoms are those of uremia.

DISEASES OF THE DIGESTIVE TRACT.

AFFECTIONS OF THE MOUTH AND THROAT.

It is a time-honored custom to begin the examination of a patient by inspecting his tongue, and if the other structures about the mouth, the lips, teeth, gums, fauces and pharynx, are included in the inspection, much of diagnostic importance can thus be elicited.

The patient should face a good light, or if the latter is artificial it should be reflected into the mouth. The examiner should first inspect the *lips*, noting their color—blue in cyanosis, pale in anemia, dry and often brown in fevers—and the presence of any fissures or ulcers or of herpes. Then notice the *teeth*, whether they are moist, or dry and covered with sordes; whether they are decayed or many missing, this tending to bring about various digestive and other disturbances; and if the patient is a child, whether the proper number of teeth have appeared, and whether they are notched (hereditary syphilis). Note the color of the *gums*, and whether they are spongy and soft, indicating scurvy. Look for the blue line near the insertion of the teeth which indicates lead poisoning.

Then have the patient protrude his *tongue*, and note whether he puts it out in a straight line—deviation indicates unilateral palsy. Notice whether it is tremulous, as in fevers and exhaustions. Note its color. It should be red and moist. The coated tongue of gastric disorder, the reddened tongue of fever, the brown, fissured tongue of the typhoid state, the strawberry tongue of scarlet fever, and the pigmented tongue of Addison's disease should be noted. The fissured tongue suggests kidney disease; the dry tongue, lack of digestive

activity. Note any ulcers or marks of the teeth upon the tongue.

Introducing a tongue depressor, note the color and general appearance of the soft palate, fauces, and pharynx. Note the presence of any ulcers or mucous patches. Determine whether the tonsils are enlarged, and whether there is any exudate upon them; and if there is, ascertain the condition of the underlying surface. (Notice whether the exudate appears upon the pillars of the fauces or the uvula.)

Inspect the *pharynx*. Note if it be markedly granular, if enlarged follicles are present, if there is an excess of mucus or any ulceration.

If the *breath* be bad, note whether the foul odor comes from the nose or the mouth, having the patient breathe through each in turn. If both are bad, the odor may come from the lungs, and will be increased by having the patient cough. It may be uremic (urinous odor), diabetic (sweetish), or simply the result of digestive disorder.

STOMATITIS.

Etiology.—Inflammation of the mouth is excited by *local irritation*, which may be mechanical (*e. g.*, injury from a sharp tooth), chemical (drugs and beverages), thermal (hot or cold food), or parasitic. Among the predisposing causes are cachectic states and gastro-intestinal diseases. Stomatitis occurs principally in young children.

Symptoms.—The patient suffers with pain and heat in the mouth, accompanied with an increased secretion of saliva, fetid breath, and fever. A young child becomes restless, and is disinclined to nurse.

Varieties.—1. *Simple stomatitis.* Diffuse redness and swelling of the mucous membrane; no ulceration.

2. *Aphthous stomatitis* (herpetic, vesicular). Vesicular spots which break down and become small ulcers.

3. *Ulcerative stomatitis.* Inflammation along the border of the gums which becomes ulcerative.

4. *Membranous stomatitis.* Catarrhal inflammation followed by the appearance of a pseudo-membrane, which becomes disintegrated and cast off, leaving an ulcerated surface. This may be a diphtheritic extension or the effect of a corrosive poison.

5. *Gangrenous stomatitis* (noma; cancrum oris). A small blister appears inside the cheek and extends rapidly, with induration, sloughing, and extensive tissue destruction.

6. *Mycotic stomatitis* (thrush). A fungus (saccharomyces albicans) appears in numerous milk white elevations, which leave a raw, bleeding surface when detached.

7. *Bednar's aphthæ.* Attacks only infants under six weeks of age. Small superficial ulcers on the palate, due to traumatism (nipple).

Prognosis.—Bad in the gangrenous form, grave in the membranous, guarded in the ulcerative, and in the other varieties favorable.

Treatment.—Secure cleanliness of the mouth by swabbing with boric acid solutions. In the gangrenous form the lesion must be thoroughly cauterized, antiseptics being used thereafter. Among the more important remedies are *argent. nitr.*, *borax*, *hepar*, *kali chlor.*, *mercurius*, *nitric acid*, and *sulphur.*

GLOSSITIS.

Etiology.—Inflammation of the tongue may be superficial or parenchymatous, acute or chronic. It may result from traumatism, toxic irritants, such as tobacco fumes, acids, etc., and occasionally it is symptomatic of digestive inflammations and infectious fevers.

Symptoms.—The superficial variety is accompanied by slight stiffness, soreness, and pain when the tongue is moved. The parenchymatous inflammation is, fortunately, rare; the tongue

becomes enormously swollen and may finally suppurate, acute pain and serious interference with respiration accompany it, and fever and other constitutional symptoms may be severe.

Prognosis.—The superficial form recovers quickly, but the parenchymatous variety is severe, often fatal, and at best is apt to lead to permanent deformity of the tongue.

Treatment.—The superficial form requires only local cleanliness to bring about recovery. The parenchymatous form may demand longitudinal incision of the tongue, even before suppuration, and often tracheotomy is necessary. Early in the attack *belladonna* is usually indicated, but the involvement of the deeper structures will require the use of such drugs as *mercury*, *lachesis*, *rhus tox.*, and *hepar*. Edema may suggest *apis* and in the stage of resolution the *iodides* and *sulphur* are useful. The mouth should be cleansed with solutions of boric acid, listerine, or some similar preparation.

ACUTE TONSILLITIS.

Etiology.—The exciting cause is probably an infective agent, the predisposing factors including—

1. Exposure to cold and wet.
2. Diathetic states, especially gout.
3. Acute infectious disease (measles, scarlatina, rheumatism).
4. Youth or adolescence.

Pathology.—According to the extent of the lesions three varieties are recognized, viz.:

1. *Acute catarrhal tonsillitis*, in which the inflammation is limited to the mucous membrane covering the tonsil, the latter appearing swollen and congested.

2. *Acute follicular tonsillitis*, in which the inflammatory process leads to desquamation of the epithelium and the latter collects in the crypts of the tonsil as small, whitish, cheesy masses.

3. *Acute suppurative tonsillitis* (phlegmonous tonsillitis, quinsy) in which the glandular structure becomes inflamed and undergoes suppuration.

Symptoms.—The onset is usually sudden and is marked by *sore throat*, moderate *fever*, and the usual febrile symptoms (*headache*, *backache*, *nausea* or *vomiting*, etc.). The swelling of the tonsils may cause distress on deglutition, and in some cases edema of the palate and pharynx leads to dysphagia and even serious dyspnea.

Diagnosis.—As a rule, inspection of the throat suffices for diagnosis. In *diphtheria* the exudate when stripped off shows a raw surface beneath, the exudate usually extends to the fauces or uvula, and doubt can be removed by examination of a culture for the Klebs-Lœffler bacillus.

Prognosis.—Favorable. Recovery within a week is the rule.

Treatment.—If there is much inflammation cold should be applied externally and the patient given bits of ice to suck. The catarrhal form yields quickly to *guaiacum*. The more severe cases frequently present indications for *aconite*, *belladonna*, or *phytolacca* at the outset. Following this, *mercur. biniod.* or *rhus tox.* may be required, or the tendency to suppuration may suggest *baryta carb.* or *hepar.* When pus can be made out, puncture the abscess with a sharp-pointed bistoury wrapped to within one-half inch of its point with adhesive plaster. In the intervals between recurring attacks, attention should be directed to the discovery and treatment of any diathetic state.

HYPERTROPHY OF THE TONSILS.

Etiology.—Chronic enlargement of the tonsils is particularly common in children, owing to strumous conditions, recurrent attacks of acute tonsillitis, or involvement of the tonsils in connection with the acute infectious diseases. It is often associated with adenoid overgrowths at the vault of the pharynx.

Pathology.—The lymphoid elements may be multiplied, the gland being enlarged but soft; or the fibrous tissues may be increased, rendering it firm in consistence.

Symptoms.—The majority of the symptoms are the result of mechanical interference with respiration; they include *mouth-breathing*, *snoring*, disturbed rest, and even attacks of dyspnea. The voice is thick and nasal, and hearing may become impaired through implication of the eustachian tube and middle ear. As the result of deficient æration of the blood, general nutrition is poor, the patient is pale and anemic, his face takes on a vacant expression, and the chest may become actually deformed into a "chicken breast."

Diagnosis.—By examination of the throat. Palpate the tonsil to decide whether the hypertrophy is fibrous.

Prognosis.—While the hypertrophy tends to disappear about the time of puberty, much damage to the child's development may have been done by that time. The prognosis as to the latter depends, therefore, upon the time of removal of the cause.

Treatment.—If the tonsil is hard and fibrous, tonsillotomy is advisable; but if the tissues are soft improvement can often be secured by the persistent administration of *mercur. biniod.*, or the *iodides of iron*, *lime*, *sulphur*, etc.

ACUTE CATARRHAL PHARYNGITIS.

(Acute Sore Throat.)

Etiology.—Acute inflammation of the mucous membrane of the pharynx occurs, especially in children, as the result of exposure to cold and wet. Diathetic states predispose to it.

Symptoms.—The throat becomes *raw* and *sore* and there is *pain* on swallowing, with a constant inclination to clear the throat. Extension to the eustachian tube may produce partial *deafness*, or involvement of the larynx may cause *hoarseness*. Examination shows the pharynx to be red and often covered with mucus.

Treatment.—Gargles of hot water, inhalations of steam, applications of pure glycerine or astringents, and sprays of albolene are useful aids in relieving the sore throat. The more important internal medicines are *aconite*, *belladonna*, *guaiacum*, and *phytolacca;* occasionally indications exist for *apis* (edema), *lachesis* (hyperesthesia), etc.

CHRONIC PHARYNGITIS.

Etiology.—Chronic pharyngitis usually occurs in adults as the result of repeated acute attacks, of the use of alcohol or tobacco, of long-continued nasal catarrh, or of over-use of the voice.

Varieties.—1. *Hypertrophic pharyngitis* (granular or follicular), in which the mucous membrane is congested, thickened, traversed by dilated blood vessels, and studded with the "split-pea" elevations of distended follicles.

2. *Atrophic pharyngitis* (pharyngitis sicca) in which the mucous membrane is pale and dry, while secretion is scanty.

3. *Ulcerative pharyngitis.* Ulceration of the pharynx may be due to syphilis, tuberculosis, diphtheria, simple inflammation, or lowered nutrition. That due to tuberculosis is painful, while the syphilitic is painless and may be associated with mucous patches.

4. *Phlegmonous pharyngitis*, *i. e.*, suppuration involving the pharynx, may be due to extension of a tonsillitis, to acute infection of the pharynx itself, or to injuries, scalds, or corrosive poisons.

Symptoms.—In all these varieties there are sore throat, weak or husky voice, and disagreeable sensations of fulness, tickling, or even pain. The phlegmonous form is attended with high fever, difficult deglutition, and rapid prostration, and may end fatally.

Treatment.—This is usually directed to the discovery and removal of the cause. Enlarged follicles are removed by the galvano-cautery, ulcers are treated with silver nitrate and

antiseptics, and constitutional states, such as those associated with tuberculosis or syphilis, receive appropriate treatment.

RETROPHARYNGEAL ACSCESS.

Retropharyngeal abscess, a suppurative process occurring in the connective tissue between the pharynx and the vertebral column, is the result of caries of the vertebræ, tubercular or syphilitic, of naso-pharyngeal disease, of the acute infections, or of faulty nutrition. It is accompanied by *pain*, *swelling*, interference with respiration and deglutition, stiffness of the neck, sometimes hoarseness, and *a doughy tumor in the posterior wall of the pharynx* which can be detected with the finger before it can be seen. It should be incised in the median line at once.

ACUTE ESOPHAGITIS

Occurs practically only as the result of swallowing hot or corrosive liquids. It is marked by pain beneath the sternum, especially on swallowing; or in severe cases deglutition may become impossible. The treatment is limited to swallowing bits of ice or bland drinks, the use of nutrient enemata, and the administration of such remedies as *arsenic*, *belladonna*, *mercury*, and *rhus tox*.

STENOSIS OF THE ESOPHAGUS.

Etiology.—The obstruction may be:

1. *Spasmodic*, occurring in hysterical patients, and in hydrophobia, chorea, and epilepsy.

2. *Organic*, due to the pressure of a tumor, sometimes an aneurism, on the esophagus, to a neoplasm (usually carcinoma) of the esophagus itself, to the presence of foreign bodies, or to a cicatricial narrowing of the tube following ulceration (corrosive poison or syphilis).

Symptoms.—Difficuty in deglutition, regurgitation of food, and dilatation of the esophagus above the point of obstruction may be noted.

Diagnosis.—A *spasmodic stricture* is usually paroxysmal, is associated with neurotic symptoms, and is accompanied with little or no nutritional failure. A bougie may be stopped at the seat of spasm, but in a few moments it can be passed on without the use of force. An *organic stricture*, on the other hand, is accompanied by a history of esophageal ulceration, which has been followed by the gradual appearance and persistence of the stenosis, and the obstruction to the passage of the bougie is permanent. The latter instrument must never be introduced until the possibility of an aneurism has been excluded.

Treatment.—The treatment of spasmodic stenosis is that of the causal disease. Organic stricture is, however, a serious and often fatal malady. If the cause is not cancerous or aneurismal, dilatation with graduated sounds should be attempted.

DISEASES OF THE STOMACH AND INTESTINES.

Disorder of the stomach is usually attended by symptoms referable to that organ, and especially in connection with the ingestion of food. The patient complains of actual pain or more often *disagreeable sensations in the epigastric region*, and there may be weight and burning after meals, flatulence, eructations, nausea, or vomiting.

Disordered appetite is common, and a bad taste in the mouth is remarked, especially in connection with the acute, often gouty, gastritis popularly known as "biliousness." *Thirst* may be a symptom of gastric disease, but it is also associated with diabetes, and any rapid loss of fluids such as occurs by vomiting, purging, hemorrhage, or by evaporation in fever.

Mental depression, *headache*, and *vertigo*, as well as palpitation and hiccough, are frequently the result of disordered

digestion. The presence of constipation or diarrhea may indicate that the intestines are involved in the disease. Disorder of the bowel may also be made evident by such symptoms as colicky pain, flatulent distention of the abdomen, and various changes in the character of the stools.

In some cases impaired nutrition is the only symptom of stomach or intestinal disease.

Interrogation of the Patient.—Should the symptoms elicited by general interrogation suggest disease of the gastro-intestinal tract, inquire particularly in regard to:

1. *Appetite.*—Is it diminished or lost, or is it excessive? Does it disappear after a few mouthfuls of food? Does he become hungry after a meal? Does he suffer with thirst?

2. *Meals.*—What is their arrangement and character? Does he eat between meals?

3. *Abnormal Sensations.*—What is their nature, and exactly where are they felt? Are they produced or relieved by taking food? How long after eating do they come on? Are they influenced by the kind of food?

4. *Belching.*—Is there much belching, and if so, when? Is it after food only, or between meals? Has the gas any odor?

5. *Regurgitation.*—Does the food ever come up into the mouth? If so, is it sour or hot? How frequently does it occur, and how long after meals?

6. *Vomiting.*—How often does it occur? Is it soon after a meal, or at other times? Does it ever occur at night? Does it relieve pain or not? Does he strain or retch much, or does it come up easily? What is the amount, color, and taste of the vomited matter? Does it smell bad? Was there ever any blood (either red blood or "coffee-grounds") in it?

7. *Bowels.*—How often are they opened? What is the character of the stools? Is there much passing of gas?

8. *Diarrhea.*—What is its frequency? What is its relation to meals and to special articles of food? Do the stools ever contain blood or slime? Does he strain during defecation?

9. *Constipation.*—Is it habitual? How often are the bowels opened? Is there any grooving or flattening of the stools? Is there any griping pain? Does constipation alternate with diarrhea?

10. *Pain* in the abdomen. Is it intermittent or continuous? Where is it felt worse? Is it aggravated or relieved by pressure?

Examination of the Patient.—Having completed the interrogation of the patient, the examination should *begin with a thorough investigation of his heart, lungs, and kidneys;* for in a large proportion of cases digestive disorder is secondary to diseases of those organs. This should be followed by an investigation directed to the gastro-intestinal system itself, beginning with:

External Examination.—Note the general appearance of the patient; emaciation and cachexia are important. Inspect the oral cavity; a coated tongue, bad teeth, an elongated uvula, or a pharyngeal catarrh may bear an important relationship to gastric symptoms. The contour of the stomach, its displacement, or its association with a tumor, is often discovered by simple inspection of the abdomen. Frequently it is advisable to examine the rectum through a speculum.

Palpate the abdomen with the tips of the fingers while the patient lies at ease; this will disclose points of localized sensitiveness and of resistance. The position of abdominal organs other than the stomach is determined by the left hand pushing the organ toward the palpating right hand; in this way the spleen or liver, if enlarged, and the kidney, if displaced, can be made out. Palpation of the abdomen with the patient in the knee-chest position is sometimes of great value. Intestinal symptoms frequently demand that the inquiry be extended to the rectum. Digital examination of the latter should be made with the patient in different positions, and in certain cases—for instance, when there is reason to suspect a malignant growth high up—the whole hand may be introduced while the patient is under the influence of a general anesthetic.

Percuss lightly to determine the position of the stomach. As it and the adjacent intestines are generally filled with air, it has been advised by some clinicians that the patient drink a large quantity (a quart) of water, so that the resulting dulness can be outlined. Another method, often objectionable, is that of administering a small teaspoonful of tartaric acid in solution and immediately afterwards a similar quantity of sodium bicarbonate, so that the resulting gas will balloon the stomach. The best practical method, however, is to insert a tube with an attached bulb, by means of which air can be forced in and the stomach distended. Auscultatory percussion may be used; the stethoscope is placed in the angle between the xiphoid cartilage and the left costal margin, and the examiner after determining the note over the stomach by light percussion, begins at the middle of the abdomen and percusses upward toward the umbilicus until a note similar to the first is heard. This may be a valuable aid in mapping out the organ.

Otherwise, auscultation is of little value in stomach examinations. The deglutition sounds heard at the ensiform cartilage evidence the permeability of the cardia. Over the intestines auscultation is used to determine the presence of peristaltic movements; absence of the normal gurgling sounds indicates paralysis of that part of the bowel, and if their absence is general, the complete intestinal paresis of diffuse peritonitis is undoubtedly present.

Examination of the Stomach Functions.—In many cases interrogation of the patient, aided by the external examination, will afford sufficient information for a correct diagnosis. If any doubt remains, however, it is essential to investigate the secretory, absorbent, and motor functions of the stomach.

Secretory functions.—As an aid in determining the secretory activity of the stomach, it is customary to administer a test meal (generally a test breakfast), consisting of one unbuttered roll or a slice of bread, and a cup of water or weak tea. One hour later this is removed through a tube and examined macroscopically and chemically. For this purpose, elaborate

quantitative analyses may be made, but practical results are obtained in a few minutes by the method here outlined.

Withdrawal of the Stomach Contents.—The patient is seated in a chair with a towel or apron about his neck, and he holds a wide-mouth bottle in his hand. The soft rubber stomach tube, about a yard in length, has several openings at the lower end and a small glass tube at the upper end. The physician dips this rubber tube in warm water, places the glass end in the bottle, tells the patient to open his mouth, and inserts the tube to the pharynx. Then the patient is told to swallow once or twice, and the tube is quickly pushed into the stomach. The desire to gag is overcome if the patient takes deep breaths. A towel wrapped around the tube will prevent saliva from running into the receptacle.

The contents of the stomach may then be withdrawn by aspiration, a rubber bulb being attached to the tube; but as a rule it is enough to have the patient take a deep inspiration and then compress his abdominal walls in the same manner as during defecation. The pressure thus exerted upon the gastric contents expels them through the tube into the bottle. When removing the tube, close the glass opening with the finger, lest some of the contained particles slip into the trachea.

The fluid withdrawn contains a sediment of partially digested bread. If fermentation is occurring in the stomach there will be a layer of foam on the surface and an odor of butyric acid. If any considerable quantity of mucus is present, it can be partly lifted from the surface with a glass rod.

Chemical Examination of the Stomach Contents.—A small quantity of the contents is allowed to pass through filter paper. A porcelain dish is placed under a Mohr's burette (50 c.c.), the latter being filled with a decinormal soda solution.

Test 1. Free Hydrochloric Acid.—Place 5 c.c. of the filtered stomach contents in the dish, together with a drop or two of dimethyl-amido-azo-benzol (½ of one per cent. alcoholic solution). If this becomes pink, free hydrochloric acid is present.

Add the soda solution drop by drop until the hydrochloric acid is neutralized and the yellow color returns; the number of c.c. of soda solution used indicates the amount of hydrochloric acid present.

Test 2. Organic Acids.—To the same 5 c.c. of contents add a few drops of a congo red solution (1%); if any further free acid is present, this turns violet. The free hydrochloric acid having already been neutralized, such free acid must be organic; its amount is estimated by the number of c.c. of soda solution necessary to restore the red color.

Test 3. Combined Acid.—Add a few drops of phenol-phtalein (1% alcoholic solution) and continue the addition of the soda solution drop by drop until the resulting pink color becomes no deeper and does not disappear on shaking. The result represents the amount of the combined acid and acid salts.

Estimation of the Acidity.—As the tests are made with 5 c.c. of stomach contents, it is necessary to multiply the results by 20 to equal the percentage of 100 c.c. This having been done, in normal cases the free hydrochloric acid (test 1) should be from 10 to 15, there should be no organic acids (test 2), and the combined acids (test 3) should be from 40 to 50. The sum of the results represents the *total acidity.*

Lactic acid.—The lactic acid forming bacilli do not grow in the presence of free hydrochloric acid (test 1); and so if the latter is present no test for lactic acid is necessary. If free hydrochloric acid is absent, however, and the congo red reaction (test 2) is positive, it is necessary to resort to:

Test 4.—Mix one drop of tincture of iron with enough water to leave a barely perceptible color, and divide the liquid between two test-tubes. Retaining one for comparison, to the other add a few drops of the filtered stomach contents; a yellow color indicates lactic acid. For delicate testing, an extract made by shaking the stomach contents with ether should be used instead of the contents themselves.

Blood.—Should blood, either by its fresh red color or in the form of brown "coffee grounds," be unrecognizable, and yet

its presence be suspected, shake up the stomach contents, allow them to settle but a few seconds, and pour off a small quantity from the top for microscopical examination; or else resort to:

Test 5.—Mix 5 c.c. of the filtered contents with 3 c.c. of glacial acetic acid and add 5 c.c. of ether. Shake well, set aside until the ether rises to the top, and pour the latter off. To it add a few drops of tincture of guaiac and a small amount of hydrogen dioxide. A dark blue color indicates the presence of blood.

Absorbent Function.—Administer 3 grains of potassium iodide in a gelatin capsule, being careful to wipe the outside clean, and then examine the saliva every minute or two for iodine. This is done by moistening strips of starch paper with the saliva, and adding a drop of fuming nitric acid; a slightly blue color indicates the presence of iodine. Normally the reaction should appear in from 8 to 15 minutes.

Motor Function.—Wash the stomach out in the morning before breakfast, the patient having eaten a hearty meal the night before; or wash it out two or three hours after the test breakfast. If any notable quantity of food is found in either case, it shows that movement through the stomach is retarded.

Signiricance of the Results.—The information secured by testing the functional activity of the stomach, while rarely pathognomonic, is of great importance in differentiating the various diseases.

1. *Changes in the quantity of hydrochloric acid* simply indicate variations in the glandular activity of the stomach. This may be functional, the result of neurotic conditions, or organic. An increase of hydrochloric acid is usually functional (hyperchlorhydria) but it may lead to organic lesions (*e. g.*, gastric ulcer). A decrease in the quantity of hydrochloric acid may also be functional (neurotic), but more often it is due to organic disease, such as cancer or chronic gastritis. A coincident excess of mucus indicates the latter. Marked variations in the quantity of hydrochloric acid indicate a nervous form of dyspepsia.

2. *The presence of organic acids* (lactic, acetic, butyric) is abnormal one hour after the ingestion of the food, and suggests either carbohydrate fermentation, due to a lack of the digestive juices (atrophic stage of chronic gastritis; cancer), or long retention, as the result of motor insufficiency (dilatation of the stomach).

3. *Persistent diminution of the total acidity*, especially if associated with absence of free hydrochloric acid, indicates glandular atrophy. If lactic acid is present, it favors a diagnosis of gastric cancer.

4. *An excessive quantity of stomach contents* (the normal return from a test meal is from 60 to 100 c.c.) indicates either excessive secretion or pyloric obstruction The latter is often evidenced by exaggerated peristaltic movements visible through the abdominal wall. Occasionally simple muscular asthenia is present, in which case the test for motor activity will afford enlightenment.

5. *Blood* may be present in the stomach contents from many causes. It may indicate the existence of a gastric ulcer, gastric cancer, acute gastritis (especially toxic gastritis), the passive congestion of the viscera secondary to heart disease or cirrhosis of the liver, or rupture of an atheromatous artery. For a complete list of the possible causes, see the etiology of *Hematemesis*.

6. The absorptive power of the stomach is decreased in cases of atrophic gastritis, cancer, and dilatation.

7. The motor activity is lessened in cases of dilatation, cancer, and the atonic form of nervous dyspepsia.

EXAMINATION OF THE FECES.

The average healthy adult passes one stool daily, but variations from two or three a day to one every two or three days are not unusual. The color and consistence of the stools also vary according to the individual, and allowance for the patient's habit must precede any deduction derived from the

character of the feces. Generally the normal stool is solid or mushy and varies in color from light yellow to dark brown or green.

Pathological stools may be changed in respect to:

1. *Consistence.*—Liquid stools characterize diarrhea and are due to rapid passage of the contents of the small intestines through the large bowel. Early frothy stools of yellow or green hue may change later in the course of diarrhea to an offensive brown fluid, or else they may become colorless and resemble "rice water," as in cholera and arsenical poisoning. The presence in liquid stools of hard round scybalæ indicates a diarrhea excited by fecal accumulation.

Firm stools, often exceedingly hard and dry, characterize constipation; sometimes they become rounded by long detention in the large bowel. They may be streaked with fresh blood from the rupture of hemorrhoidal tumors in the rectum, or tearing of the sphincter during their passage.

2. *Color.*—Diet affects the color of the stools, a milk diet causing light yellow, and one of meat leading to dark brown stools. Some drugs, such as iron and bismuth, color the stools black; calomel renders them green, and rhubarb, santonin, and senna make them yellow. Altered blood, the result of hemorrhage high up in the intestine, renders the stools black, tarry, and offensive. If the influence of food or drugs and the presence of blood or pus can be excluded, changes in the color must be attributed to alterations in the quality or the quantity of the bile present. Green stools are seen early in many diarrheas, especially those of children. Small yellowish-white masses may suggest the presence of undigested casein, but if they are dissolved by ether, they are fatty. Fatty stools, sometimes appearing like tallow, occur in connection with amyloid disease, tuberculosis, pancreatic disease, and atrophy of the intestinal mucous membrane. White, clayey stools, which also owe their color to the fat they contain, are often seen in connection with obstructive jaundice or deficient bile formation.

3. *Odor.*—The stools of breast fed infants are normally sour in smell. Infantile intestinal catarrhs may also occasion this odor; or it may become cadaverous, as a result of dysenteric, syphilitic, or cancerous diseases of the rectum. The absence of bile seems to cause an offensive odor of the stools.

4. *Abnormal Constituents.*—Blood from the rectum is bright red and streaks the surface of the stool; it may be due to hemorrhoids, or to inflammation or malignant ulceration of the rectum or colon. Blood entering the bowel higher up becomes mixed with the feces; it may be brownish-red, black and tarry, or it may resemble coffee grounds, and its presence suggests intense congestion or ulceration of the stomach or bowels. In case there is doubt as to its presence, mix a portion of feces (rubbed up with water and filtered, if necessary) with a few c.c. of glacial acetic acid, and then shake with ether. If the ether does not settle down clear, add a few drops of absolute alcohol. A bronchial red color of the layer of ether indicates the presence of hematin acetate, *i. e.*, of blood.

Pus may be recognized in the stool in exceptional cases. Ulcers of the small intestine may give rise to small grayish-white masses composed of pus cells and mucus. The rupture into the bowel of an abscess is sometimes followed by the discharge of a large quantity of pure pus.

Concretions, such as gall-stones and enteroliths, as well as foreign bodies and parasites, are best discovered by washing the feces with running water over a linen sieve.

Microscopical examination of the feces, while of limited value, may occasionally afford useful information. In addition to particles of food (muscle and elastic fibres, fat drops, vegetable cells) and the epithelial cells, blood corpuscles, crystals, and debris from the intestine itself, it may be possible to recognize parasites and bacteria. For the identification of the latter, of which the more important are the bacilli of cholera, typhoid, and tuberculosis, culture methods are often necessary.

ACUTE GASTRITIS.

Etiology.—Acute inflammation of the stomach may be—

1. The result of dietetic errors (food of improper character, excessive in amount, or too hastily swallowed; alcohol; iced drinks, etc.).

2. An accompaniment of acute infectious diseases.

Pathology.—The mucous membrane becomes red, swollen, covered with mucus, and sometimes eroded.

Symptoms.—Epigastric distress, nausea, and vomiting of food, mucus, and bile usher in the attack. There may be some pyrexia, the tongue is coated, and headache and other febrile symptoms are present. Should the stomach not succeed in emptying itself by vomiting, the intestines become involved. In consequence a diarrhea is set up, and it is accompanied with more or less abdominal pain and tenderness.

Diagnosis.—*Typhoid fever* and other oncoming infections may be simulated by febrile cases of acute gastritis, but the latter always terminates within a week. In doubtful cases, the existence of herpes labialis favors a diagnosis of gastritis; the occurrence of the diazo-reaction, typhoid.

Biliary colic is accompanied by greater pain, which is located about the gall-bladder or ducts, and jaundice is frequently present.

Prognosis.—Recovery usually occurs within five days.

Treatment.—Often the attack can be subdued immediately by placing the patient in a warm bath and adding as much hot water as can be borne; continue the bath for half an hour or more. Vomiting rids the stomach of offending food, and on that account should be encouraged; if it is necessary to aid nature, let the patient drink a pint of lukewarm water and tickle the fauces. The stomach having been emptied, permit no food for a day, and then begin feeding cautiously with milk and soups. During the acute stage small quantites of water or bits of ice may be swallowed in order to quench the thirst.

Of remedies, *apomorphia* or *ipecac* may be required to control excessive vomiting; unusual prostration will suggest *arsenic;* and *nux vomica* will be indicated by a history of dietetic errors or alcoholism. A heavily coated tongue with great nausea and pain suggests the administration of *antimon. crud.*, bilious vomiting with intense distress in the epigastrium is relieved by *iris*, *pulsatilla* is used in attacks brought on by indulgence in fats or acids, and *bryonia* in those cases which result from over-indulgence in cold drinks. *Cuprum ars.* is almost specific in cases in which intestinal involvement is indicated by the onset of colic and diarrhea.

Acute Toxic Gastritis, the intense inflammation of the stomach which follows the ingestion of irritant poisons (arsenic, acids, corrosive sublimate, etc.), is attended with severe symptoms of sudden onset, including pain, vomiting and collapse, The diagnosis is often made after questioning the patient or by detecting traces of the poison about the mouth or in the vomitus. Treatment must include the appropriate antidote, followed by rest and probably rectal feeding until the eroded stomach walls can heal.

Phlegmonous Gastritis, a rare suppurative inflammation of the stomach, occurs by metastasis in pyemic and other septic states, and sometimes it is apparently idiopathic. It is attended with severe pain, high fever, and complete anorexia, and death ensues within a week or two. Diagnosis during life is impossible.

CHRONIC CATARRHAL GASTRITIS.

Etiology.—Chronic catarrhal inflammation of the stomach may be induced by—

1. Dietetic errors.
2. Repeated attacks of acute gastritis.
3. It may be a symptom of heart, lung, liver, or kidney disease.

Pathology.—The mucous membrane is of a yellow or slate

color, sometimes presenting ecchymotic spots, and covered with mucus. Microscopically it may be seen that the disease has begun as a glandular inflammation, and this has been followed by interstitial changes. Atrophy of the mucous membrane, with complete glandular destruction, may finally ensue.

Symptoms.—The disease is of gradual development, and at first the symptoms are obscure. There may be a disagreeable taste in the mouth, bad breath, a coated tongue, deranged appetite, and a sense of epigastric fulness which at times leads to palpitation and dyspnea. Belching, vertigo, and headache are common, but pain is generally absent. Vomiting is not usual; when it occurs, it is apt to be in the morning. The bowels are constipated as a rule, and the urine is scanty and deposits urates and phosphates.

The general symptoms include languor, irritability, mental inactivity. and a desire to yawn. As a rule, the patient appears well nourished, but late in the course of the disease considerable emaciation may take place.

Gastric Contents.—Examination of the stomach contents one hour after a test meal shows lessened acidity, little or no free hydrochloric acid, and often much mucus.

Diagnosis.—The long continuance of the symptoms described, and the low acidity of the stomach contents, associated often with an excessive quantity of mucus, render the diagnosis clear.

Ulcer and *cancer* are generally distinguished by severe pain, hematemesis, and emaciation; in the case of ulcer there is a circumscribed tender spot in the epigastrium, and in cancer the tumor may be detectable.

A *gastric neurosis* may present symptoms suggestive of chronic catarrhal gastritis, but the coexisting nervous disturbances and the sudden changes in the subjective sensations and in the chemical condition of the gastric contents will aid in its recognition.

Prognosis.—The disease is of long duration, and becomes

more serious in proportion to the decrease of gastric secretion. If the condition is not secondary to some incurable disease of another organ, recovery or at least improvement is generally possible; but relapses are common. Chronic gastritis tends to complete destruction of the secreting structures (*achylia gastrica*) and dilatation of the stomach, and these changes represent an incurable condition.

Treatment.—When a patient submits himself to treatment, for the first week or two his meals must be regular, carefully masticated, and very light (milk, koumyss, oatmeal, rice and chicken soups, soft boiled eggs, mashed potatoes, scraped meat, and toasted bread). Then the amount of food must be increased gradually, and he must continue to abstain from indigestible food, alcohol, and tobacco. Rest after meals, regular hours of sleep and rest, moderate open air exercise, and a morning sponge bath should be enjoined. A tumblerful of hot water half an hour before each meal serves to clear the stomach of mucus; but in severe cases daily lavage followed by the administration of a pint of peptonized milk affords a preferable method of accomplishing this result. The drinking of a few spoonfuls of hydrogen dioxid, somewhat diluted, followed in a few moments by the ingestion of a pint of hot water, is often equally effective. It in turn should be followed by the administration of peptonized milk.

Of medicines, *nux vomica* is particularly useful in atonic, constipated patients by whom purgatives have been abused. *Hydrastis* is indicated by the presence in the stomach of excessive quantities of mucus, with vomiting, constipation, and often a "sinking" sensation in the epigastrium. *Pulsatilla* may be given in similar cases in which flatulence and eructations have been brought on by indulgence in rich food. Obstinate cases with great flatulence, vomiting of mucus, and epigastric distress are relieved by *argentum nit.* *Arsenic* is indicated by extreme gastric irritability, with burning pain, thirst, restlessness, and anxiety; while *bryonia* is suitable for cases presenting great dryness of the digestive tract, the un-

digested food causing a sense of weight in the stomach, together with constipation and liver disturbance.

Lycopodium (flatulent, lithemic patients), *sepia* (neurotic women of gouty habit), *cinchona* (patients debilitated by acute diseases), and *sulphur* (chronic gastritis associated with liver disease and hemorrhoids) are also remedies of value.

Carbolic acid in doses of 2–5 drops, well diluted, of a one per cent. watery solution, administered after cleansing the stomach, is a remedy of great value, particularly in gastric catarrhs of alcoholic origin. Great flatulence may justify the administration of *bismuth subgallate* or *sodium sulphocarbolate* in 5–10 grain doses after meals.

ULCER OF THE STOMACH.

Etiology.—A gastric ulcer may be due to:

1. Malnutrition of the mucous membrane of the stomach (anemia, chlorosis, heat, or kidney disease, etc.).

2. Local thrombosis and embolism, the resulting blood stasis permitting digestion of the membrane by the gastric juice.

3. Hyperchlorhydria.

4. Mechanical injury (by pressure, as in shoe making).

5. Predisposition (young women).

Pathology.—The digestion of a portion of the mucous membrane by the gastric juice leaves a round, funnel-shaped ulcer of varying depth. Generally it is situated on the posterior wall of the greater curvature of the stomach. Rarely the ulcers are multiple.

Course.—1. The ulcer may cicatrize. This termination, usually favorable, may nevertheless lead to contraction and stenosis at the pyloric orifice; or the cicatricial tissue may later become the seat of malignant disease.

2. The ulcer may persist for years without healing.

3. The ulcer may penetrate the stomach wall, the gastric contents entering the peritoneal cavity and causing a rapidly fatal septic peritonitis.

4. Adhesions may form between the stomach and the adjacent viscera (liver, pancreas, omentum) preventing or at least localizing the peritonitis.

5. Fistulous tracts into these adjacent viscera, or into the pleura or pericardium, may be formed.

6. Erosion of a blood vessel may occur; the gravity of the ensuing hemorrhage varies according to the size of the vessel thus opened.

Symptoms.—Many cases of gastric ulcer present no special symptoms; the lesion may be discovered only at autopsy following death from other causes. In typical cases, however, the disease is characterized by:

1. *Pain*, especially, but not necessarily, after taking food, and often relieved by vomiting.

2. *Tenderness* on pressure over the ulcer. This is strictly localized to a small spot, which is often an inch or two above the umbilicus.

3. *Vomiting*, shortly after eating, the vomitus usually containing an excess of hydrochloric acid.

4. *Hemorrhage*, which is often quite profuse and leads to vomiting of bright red blood or the passage of tarry stools.

5. *Anemia*, usually of chlorotic type, accompanied by progressive emaciation.

6. *General dyspeptic symptoms*. These are present in varying degree, and in some cases constitute the only signs of the disease.

7. *Gastric Contents*.—The stomach tube must not be used; but examination of the vomitus usually discloses great hyperacidity of the gastric juice.

Diagnosis.—In *gastralgia* the pain is irregular in its occurrence, without aggravation by food; localized tenderness, vomiting, hematemesis, and dyspeptic symptoms are absent, and the neurotic character of the patient is usually evident.

Chronic gastritis lacks the severe pain, frequent vomiting, hematemesis, and anemia, which usually characterize cases of

gastric ulcer, while the amount of hydrochloric acid in the stomach contents is decreased.

In *hyperchlorhydria* the pain is apt to appear two or three hours after the ingestion of food, and it is relieved by the administration of more food or of an alkali, such as sodium bicarbonate; waterbrash is frequently present and hematemesis does not occur.

Cancer occurs later in life; the pain is less intense but more continuous, the amount of blood vomited is comparatively small, and often the vomitus betrays the absence of hydrochloric and the presence of lactic acid. The appearance of cachexia and the discovery of a palpable tumor afford conclusive evidence as to the nature of the lesion.

Prognosis.—This must be guarded, since fatal hemorrhage or perforation is possible at any time, and relapse even after apparent recovery is not unusual. Treatment is effective in recent cases, a large proportion of which recover.

Treatment.—1. Rest. Put the patient to bed.

2. Diet. For a period of a week or ten days, severe cases require rectal feeding (peptonized milk, beef juice, or peptones, four ounces every 4–6 hours). Milder cases may be given peptonized milk by the mouth (two ounces every two hours), and beef peptone and egg albumen may be substituted gradually or in part. As the symptoms subside, thin gruels, raw eggs, well-cooked rice, and custards are permitted, but the return to solid food must be very gradual.

3. Medicines. *Atropine*, 3x, controls as by magic the severe pain of a gastric ulcer associated with hyperchlorhydria (Goodno). *Argent. nit.*, in freshly-made watery solution, is an invaluable remedy when there is epigastric pain and belching of great quantities of gas. *Arsenic* is useful when its characteristic thirst, burning pain, vomiting, and prostration are present. *Kali bich.* is suggested by catarrhal symptoms and vomiting of thick mucus, while *hydrastis* is suited to catarrhal cases with sensations of weakness and aching and cutting pains in the epigastrium, together with loss of appetite. The

distinct etiologic relationship of hyperchlorhydria to ulcer of the stomach renders the treatment advised for the former condition equally applicable to many cases of gastric ulcer.

Hemorrhage may be met by the use of ice externally and internally, small bits being swallowed by the patient, and the administration of *ipecac*, *hamamelis*, or *hydrastinine hydrochlorate*. Severe hemorrhage or symptoms of perforation may require *morphia*, gr. ¼, hypodermatically; and at times surgical intervention is advisable.

CANCER OF THE STOMACH.

Etiology.—Heredity, mature life, gastric catarrh, and ulcer are among the conditions which predispose to cancer.

Pathology.—Excepting the female generative organs, the stomach is the organ most frequently attacked by primary cancer; rarely it is secondary. The growth originates in the mucous coat; any variety may occur, but the more frequent are the cylinder-celled epithelioma, medullary, scirrhus, and colloid. It may appear as a projecting tumor, a diffuse inflammation, or an annular constricting band, situated either at the pylorus (60%), the cardia (10%), or over the greater or lesser curvature. The location of the growth affects the shape of the stomach, dilatation following obstruction at the pylorus, or contraction resulting from stenosis at the cardia. The increased weight of the stomach and its adhesions to adjacent organs may serve to displace it.

Symptoms.—These are often obscure, but as a rule they develop somewhat in the following order:

1. *Dyspeptic symptoms*, perhaps with sudden loss of appetite.

2. *Failure of general health*, with increasing weakness and emaciation; finally cachexia.

3. *Epigastric pain*, constant and increasing in severity.

4. *Vomiting*, often of retained food which has become offensive, and which may be admixed with blood in small quantities.

The position of the growth may occasion some variation in the symptoms, *e. g.*, a cancer at the cardia causes pain and difficulty in swallowing.

Stomach contents. Chemical examination of the vomitus reveals absence or a minimum amount of hydrochloric acid, and lactic acid is often present. Washing out the stomach in the morning will reveal the presence of undigested food from the meals of the previous day, indicating pyloric stenosis. Microscopical examination may reveal a small amount of blood or particles of the malignant growth.

Physical Examination.—The tumor is palpable when situated at the pylorus or on the anterior wall or greater curvature. Metastatic involvement of glands, nodular infiltration of the liver or of the thoracic viscera, with indications of pleurisy, may be noted. A cancer situated at the cardia often leads to stenosis of the esophagus, with absence of the deglutition sounds at that point.

Clinical Course.—1. Generally, death from exhaustion ensues within two years.

2. The disease, particularly in the aged, may run a latent course, death finally resulting from some intercurrent disorder.

3. The gastric condition may be masked, the symptoms suggesting the possibility of pernicious anemia or nephritis.

4. Secondary growths of the liver or peritoneum may obscure the primary lesion.

Diagnosis.—The diagnosis of cancer of the stomach is fraught with difficulty; almost every symptom may be present, and yet be due to other causes, and in other cases characteristic signs of the disease may be utterly lacking, only autopsy revealing the presence of the neoplasm. The symptoms and signs distinguishing cancer from other gastric diseases have been considered in connection with ulcer of the stomach.

Abdominal *tumors not originating in the stomach* can be distinguished by careful physical examination as to their attachments.

Benign tumors of the stomach are rare, and their presence give rise to no malignant symptoms.

Dilatation of the stomach due to benign stenosis of the pylorus affords a history of long illness interrupted by periods of almost complete freedom from symptoms.

Prognosis.—Hopeless; death almost inevitably occurs within three years.

Treatment.—Nourish the patient with easily assimilable food. Palliate the pain with *morphia* or *svapnia*. Control vomiting with ice, champagne, and appropriate medicines. Lavage may be used to clear the stomach of irritating undigested food. When stenosis becomes extreme it is necessary to resort to rectal feeding.

Surgical interference, though hazardous, may be considered.

DILATATION OF THE STOMACH.

(Gastrectasis.)

Etiology.—The capacity of the stomach may become increased as the result of:

1. Pyloric obstruction (pyloric tumors or cicatrices, pressure on the pylorus by tumors of adjacent organs).

2. Muscular atony, due to overloading the stomach with food and drink, to gastric catarrh, or to diseases producing general atony (diseases of heart, lungs, kidneys, and blood).

Pathology.—The stomach, normally capable of containing about three pints, becomes dilated to two or three times that capacity, with thinning of the muscles and the changes in the mucous membrane characteristic of chronic gastric catarrh.

Symptoms.—The patient presents symptoms of gastric catarrh, sometimes combined with those of the causative condition (*e. g.*, tumor). In addition he vomits at intervals large quantities of undigested and fermenting food, which have evidently been accumulated within the stomach. If the vomitus is allowed to stand, a sediment of undigested food is thrown down, and microscopic examination reveals the presence in it of yeast

fungi; sarcinæ, and bacteria. Hydrochloric acid is generally replaced by lactic, butyric, and other organic acids, and the motor power of the stomach is lessened.

In advanced cases general nutrition fails, the bowels become constipated and the urine scanty, anemia and emaciation are progressive, and death finally occurs from exhaustion or toxic absorption. Cessation of the vomiting, indicating muscular failure of the stomach, is often a danger signal.

Physical Signs.—Inspection may reveal the outline of the bulging organ, palpation confirming the discovery. Percussion of the artificially distended stomach, the patient standing upright, will show its excursion below the umbilicus.

Prognosis.—This depends upon the cause. If the condition is due to cicatricial contraction or tumor of the pylorus, it is incurable, although persistent palliation is still possible; if it is the result of simple muscular atony, the prospect is fairly favorable.

Treatment.—The patient should be restricted to a dry diet, consisting of grated meats, cereals, dry bread thoroughly masticated, and prepared foods in powder form, such as somatose. If liquids are permitted at all, very small quantities should be taken at frequent intervals. Fats must be forbidden, and starch and sugar should be taken most sparingly.

Cleanse the stomach by daily lavage with a mildly alkaline solution, followed by the ingestion of a little peptonized milk. *Avoid constipation;* use enemata or laxatives if necessary.

Medicines may be given in accordance with any symptomatic indications present; their effects are necessarily limited to a control of the associated gastric catarrh, and the more important drugs are those recommended for that disease.

HEMATEMESIS.

Etiology.—Hemorrhage into the stomach, from which hematemesis usually results, may be due to:

1. *Traumatism* (mechanical injuries, such as blows or wounds; chemical injuries by acids or alkalies, etc.).

2. *Gastric diseases* (ulcer; cancer; disease of the gastric blood vessels, including atheroma and varicose veins; and gastric congestion, in connection with acute gastritis, cirrhosis of the liver, chronic heart and lung diseases, etc.).

3. *Blood disorders* (acute infectious diseases, such as yellow fever, smallpox, malaria, etc.; toxemias, such as that induced by phosphorus-poisoning; and hemorrhagic diseases, such as scurvy, purpura, and hemophilia).

4. *Nervous diseases* (hysteria, epilepsy, paresis) rarely.

5. *Melæna neonatorum*, a profuse and fatal hemorrhage in the newborn.

6. *Rupture of an aneurism* of the abdominal aorta into the stomach.

Diagnosis.—It is essential to recognize the cause of hemorrhage into the stomach, and, moreover, it is necessary to distinguish the latter from blood swallowed during an attack of epistaxis or hemoptysis and later vomited or passed with the stool.

Prognosis.—The hemorrhage is seldom immediately fatal; in other respects the prognosis depends upon the cause.

Treatment.—Rest, abstention from food, and the use of ice locally are advisable. Administer such remedies as *ipecac*, *hamamelis*, *hydrastinine hydrochlorate*, *turpentine*, or various other medicines selected according to the cause and the associated conditions. Intravenous saline infusions may become necessary in extreme cases.

GASTRIC NEUROSES.

Etiology.—Functional disorders of the stomach, due to nervous influences, are the result of

1. A neurotic temperament, hereditary or acquired.

2. Neurasthenia, due to overwork, injudicious dieting, etc.

Clinical Varieties.—Disordered innervation of the stomach may manifest itself in—

1. Neuroses of sensation (nervous dyspepsia, gastralgia, etc.).

2. Neuroses of secretion (hyperchlorhydria, occasionally hypochlorhydria or sub-acidity).

3. Neuroses of motility (increased or diminished peristalsis).

These three varieties of gastric disorder resulting from disturbed innervation are not always separated one from another by sharp lines of demarcation, and in consequence they are usually classified together under the general term of "nervous dyspepsia." In certain cases, however, notably that of hyperchlorhydria, the neurosis presents a distinct clinical type.

NERVOUS DYSPEPSIA.

(Gastric Neurasthenia.)

Symptoms.—In this form of dyspepsia the stomach is able to accomplish digestion within the normal seven hour limit, but the process is attended with annoying symptoms. The latter may be *gastro-enteric* (fulness after eating, belching of tasteless gas, nausea but rarely vomiting, rumbling in the bowels, and constipation), but they are usually associated with many *neurotic* symptoms, such as palpitation, numbness of the extremities, and anxiety. In certain cases these symptoms are associated with muscular atony of the stomach and consequent stasis of the gastric contents (*atonic dyspepsia*).

Diagnosis.—The recognition of nervous dyspepsia depends upon the *exclusion of all organic causes*, gastric or otherwise, for the symptoms. The neurotic association, together with the liability of the symptoms to appear and disappear suddenly, is an aid to diagnosis.

Prognosis.—For life, good; for recovery, doubtful.

Treatment.—That of neurasthenia or hysteria: Rest, proper exercise, a full nutritious diet, and such remedies as *argent. nitr.*, *cinchona*, *ignatia*, *lycopodium*, *nux vomica*, *pulsatilla*, etc.

HYPERCHLORHYDRIA.

Etiology.—Hyperacidity of the gastric juice may be paroxysmal, or it may be continuous (*Reichmann's disease*). It occurs particularly in neurasthenic and gouty adults as a result of emotional causes, such as overwork and worry.

Symptoms.—In the *paroxysmal variety* the patient suffers distress and pain in the stomach two or three hours after eating, with thirst, pyrosis, eructations, and even vomiting. Relief can always be secured by taking food, alkalies, or large quantities of water.

If uncontrolled, these symptoms tend to pass into the *continuous form*, in which the distressing sensations persist without intermission and are even more severe. The irritation leads to increased muscular activity of the stomach, the food being hurried on without opportunity for digestion or absorption, and ultimately in spite of the large quantity of food taken, anemia and emaciation ensue.

Course.—Hyperchlorhydria may lead to dilatation of the stomach, either through the irritating gastric juice causing spasm of the pylorus, or through atony of the weary muscular coat permitting accumulation within the organ. Increasing dilatation may ultimately lead to glandular atrophy. Ulcer of the stomach is an exceedingly common complication or sequence of long-continued hyperacidity.

Diagnosis is possible only after an examination of the gastric contents. One hour after the test breakfast the contents show an excess of hydrochloric acid, often amounting to two or three times the normal amount.

Prognosis.—The prognosis under proper treatment is favorable, except in those protracted cases in which the condition has become continuous; the latter are rarely cured.

Treatment.—The distress attending the attack can be relieved by the administration of nitrogenous food (meat and milk) or by neutralizing the acid with an alkali (sodium bicarbonate or Carlsbad salts, gr. x, every 10 minutes until relief is obtained).

In order to radically cure the disease, however, it is necessary to regulate the habits of the patient, prohibiting overwork and securing for him necessary rest and out-door exercise. The diet must consist largely of meat, carbo-hydrates being reduced in quantity to a minimum; and all substances tending to excite the activity of the stomach glands, such as spices, fruits, and acids, must be prohibited.

Of remedies, *atropine* 3x given just before each meal usually ensures almost immediate relief. *Anacardium* is often indicated by the constant hunger, with relief from eating, but aggravation in two or three hours. *Robinia*, *cuprum ars.*, *nux vomica*, and *iris* are also extremely useful in appropriate cases.

GASTRALGIA

(Gastrodynia; Cardialgia; Neuralgia of the Stomach)

Is a term properly applicable only to paroxysmal stomach pain unassociated with any discoverable organic lesion. It occurs particularly in neurotic and anemic women. For the exclusion of gastric ulcer and cancer, angina pectoris, the gastric iris of locomotor ataxia, gall-stone and intestinal colics, etc., careful and painstaking examination is often necessary. The **treatment** of the attack should include the application of external heat, the drinking of large quantities of hot water, and the administration of such medicines as *arsenic*, *belladonna*, *copper arsenite*, *iris*, and *nux vomica*.

SPLANCHNOPTOSIS.

(Glénard's Disease.)

Varieties.—Splanchnoptosis (visceroptosis) is a general term for the downward displacement of certain of the abdominal viscera, especially the stomach (*gastroptosis*), the intestines (*enteroptosis*), the kidney (*nephroptosis*), the spleen (*splenoptosis*), and the liver (*hepatoptosis*).

Etiology.—The displacement is the result of:

1. Relaxation of the abdominal walls; and

2. Loosening or overstretching of the supporting ligaments.

The condition is usually found in young women. It has been attributed to undue exertion, to injuries, and to relaxation of the abdominal walls following repeated pregnancies. The gastro-intestinal prolapse leads to a certain degree of stenosis, whereby the passage of food is interfered with and nutrition suffers in consequence.

Symptoms.—The patient suffers with a general *asthenia;* she readily becomes tired, and is comfortable only when lying down. She complains of a sense of *weight* and a sickening *dragging* in the abdomen. *Gastric symptoms* are prominent—anorexia, distress, burning, or pain in the epigastric region, nausea, vomiting, diarrhea, or constipation. The symptoms are more or less *relieved by pressure*, either by the hands or a belt, upon the lower abdomen.

Diagnosis.—In the presence of these symptoms, careful physical examination of the abdomen, made while the patient stands erect and again when she lies down, becomes necessary.

Among the objective signs the following are more or less characteristic:

1. A splashing sound (*chapotement epigastrique*) in the stomach from two to six hours after meals.

2. Pulsation of the abdominal aorta (*battement aortique*).

3. A cord-like body lying transversely at or below the level of the umbilicus, supposed to be the transverse colon (pancreas?).

4. Displaced or movable abdominal organs, especially a floating kidney.

Treatment.—The patient should wear an abdominal bandage reaching to the navel and exerting pressure upward, or, better, a special truss may be constructed for the purpose. The bowels should be kept open by means of aperients (Hunyadi, Rubinat, or Carlsbad water) if necessary. The tonicity of the abdominal walls may be improved by massage, electricity, hydrotherapy, and sometimes gymnastic exercises. Medicines

should be prescribed for the dyspeptic and other symptoms as they present themselves.

ACUTE ENTERITIS.

Etiology.—Acute intestinal catarrh is usually the result of the action of some irritant; the disease may be *primary*, due to—

1. Dietetic errors. Unripe fruit, the ingestion of food in too large quantities, and food decomposed as the result of bacterial activity.

2. Toxic causes, *e. g.*, metallic poisons and drastic cathartics.

The intestinal inflammation may be *secondary* to—

1. Gastric disorders which permit irritating substances to pass into the intestine.

2. Organic intestinal disorders, such as ulcer, cancer, hernia, and peritonitis.

3. Congestion of the intestinal blood vessels, the result of portal obstruction, occurring in chronic heart and lung diseases.

4. Acute infectious diseases, such as typhoid.

5. Chronic cachexias (nephritis, tuberculosis, cancer, anemia).

6. Foreign bodies, such as enteroliths, gall-stones, and parasites.

Pathology.—The mucous membrane becomes hyperemic and swollen, and its surface is covered with mucus, desquamated epithelium, or even pus. The process may be localized or diffuse, and may be accompanied by ulcerative changes in the glands and erosion of the mucous membrane.

Symptoms.—1. *Diarrhea*, due to increased peristalsis and augmented secretion, is the principal symptom of enteritis. It may, however, be absent if the inflammation remains confined to the upper bowel, and the colon is not involved. The early bowel movements dispose of the solid masses, and as a result the later stools become thin and finally watery.

2. *Pain.* Defecation may be preceded by colicky pain in the abdomen and followed by tenesmus.

3. Among the *associated symptoms* may be nausea, loss of appetite, great thirst, abdominal tenderness, and concentrated, sometimes albuminous, urine.

Diagnosis.—Generally the symptoms suffice for diagnosis; but as it is essential to know whether the enteritis is primary or secondary, careful examination for the probable causes of the latter is necessary.

Exact localization of the intestinal lesion is not always possible. Duodenal catarrh may be indicated by tenderness in the right hypochondrium following an attack of gastritis; the stools usually contain an excess of mucus, and occasionally jaundice is present. Catarrh of the small intestine is accompanied by an excess of indican in the urine; or it may be indicated by Rosenbach's reaction. The latter is obtained by boiling urine in a test-tube while adding nitric acid drop by drop; a burgundy-red color which persists on further boiling affords positive evidence of disturbed enteric metabolism. Catarrh of the large intestine is accompanied by tenesmus and severe colicky pains in the left iliac fossa, together with itching and burning at the anus, the passage of mucus and blood, and great pain on digital examination.

Prognosis.—Primary cases are serious only at the extremes of life; the prognoses of secondary cases vary according to their cause.

Treatment.—Rest in bed, with enforced use of the bed pan, and a light diet, are essential details in the treatment of every case. Acute catarrh of the small intestine with little associated pain is usually controlled by *bryonia*, *croton tig.*, *cinchona*, *colchicum*, or *podophyllum;* great pain indicates the need of *colocynth*, *cuprum ars.*, or *nux vomica;* and involvement of the colon suggests the administration of *aloes* or *mercurius dulcis*. Attacks resulting from improper diet, with various associated gastric symptoms, may require the use of *antimon. crud.*, *ipec.*, or *nux vomica*. *Arsenic* may prove useful in severe cases with great thirst, restlessness, etc.

CHRONIC ENTERITIS.

Etiology.—Chronic catarrhal inflammation of the intestine is usually secondary to repeated acute attacks or the persistence of the causes therefor.

Pathology.—The process consists of a chronic catarrhal inflammation of the intestinal mucous membrane with ensuing hypertrophies, atrophies, and ulcerations.

Symptoms.—1. *Diarrhea.* This is not invariable, constipation, or alternating constipation and diarrhea, being present in many cases.

2. *Mucus in the stools*, in sago-like flakes or jelly-like masses.

3. *Blood in the stools*, when ulceration occurs.

4. *Pus in the stools.* This appears when inflammation of the rectum (proctitis) has resulted in abscess-formation. A rectal fistula may ensue.

5. *Nutritional disturbances.* Emaciation and weakness are prone to occur in connection with advanced changes in the mucous membrane. Should an atrophic process involve both the stomach and bowels, a pernicious form of anemia develops.

Diagnosis.—The passage of large amounts of mucus with the stools determines the condition, and its localization may be judged according to the signs referred to in connection with the diagnosis of acute enteritis. Recognition of the primary cause is essential.

Prognosis.—Under any circumstances the prognosis as to recovery must be guarded; but it varies somewhat according to the cause and its removability.

Treatment.—1. Discover and treat the cause (heart and kidney diseases, etc.)

2. Diet. Food containing a minimum of waste, *i. e.*, milk and later nitrogenous substances should be given. For gouty and nephritic patients the latter are contra-indicated, however, and in such cases recourse must be had to stale bread and well-cooked cereals.

3. Daily irrigation of the colon with pure water, or a saline solution (2–3 pints), is advisable in obstinate cases involving that portion of the bowel.

4. Medicines must be carefully selected after consideration of the cause as well as the obtrusive symptoms. Among those most frequently indicated are *aloes*, *argent. nit.*, *arsenic*, *cinchona*, *mercur. cor.*, and *sulphur*. Intestinal antiseptics, *e. g.*, bismuth subgallate, beta-naphtol bismuth, and sodium sulphocarbolate, in doses of from five to ten grains three times a day, may be used in some cases.

CHOLERA MORBUS.

(Cholera Nostras, Sporadic Cholera)

Cholera morbus is a severe form of acute gastro-enteritis occurring particularly in hot weather and in young adults. It is due to dietetic errors followed by decomposition of the gastro-intestinal contents and toxic absorption. The attack is characterized by the sudden onset of violent vomiting, purging, and pain; and severe cases closely resemble Asiatic cholera. The treatment should be that directed for the latter disease.

THE DIARRHEAS OF CHILDREN.

Etiology.—1. Dietetic errors. The disorder is apt to occur in artificially-fed children because of improper or contaminated food.

2. Hot weather. This tends to aid bacterial development in the food, and to weaken the resistance of the child.

3. Dentition and faulty hygiene act as predisposing causes.

Pathology.—The condition varies from a simple non-inflammatory catarrh of the small intestine (*intestinal indigestion*) to an inflammation which especially affects the lymph follicles of the ileum and colon (*entero-colitis*) or an intense gastro-enteritis of bacterial origin (*cholera infantum*).

Symptoms.—These varieties are recognizable clinically, viz.,

1. *Intestinal indigestion*, characterized by slight fever, restlessness, and some purging. The stools are sour and offensive; at first they contain coagulated milk or other undigested food, but later they become greenish and watery, and often contain a little mucus or blood. The disorder may be acute or chronic; if the latter, it may lead to death from exhaustion.

2. *Entero-colitis*, the variety in which the inflammation especially affects the lymph follicles of the ileum and colon, is gradual in onset and at first suggests intestinal indigestion; speedily, however, it becomes severe, and is accompanied by high fever (104°), a distended and painful abdomen, and irritating stools containing large quantities of mucus. The child emaciates rapidly, and if the disease remains unchecked death occurs in a few weeks.

3. *Cholera infantum*, an intense gastro-enteritis due to bacterial contamination of the food, is characterized by the sudden onset of severe vomiting and purging, with high fever (105°–106°), rapid exhaustion, and often death within a few hours.

Diagnosis.—In *intestinal indigestion* the stools are at first large and contain an abundance of undigested material and a small amount of mucus. Should the disease merge into an *entero-colitis*, the stool, still large, comes to consist largely of mucus and serum and a characteristic cadaveric odor is present. The child passes, perhaps, eight or ten stools daily; they tend to become smaller and more frequent, and much rectal irritation is present. If the mucus is intimately mixed with bile, its origin is in the small intestine; if it is clear or only streaked with bile, it comes from the lower bowel. Dark blood comes from high up in the intestine; bright blood, from the lowel bowel; masses of jelly-like mucus, from the rectum or lower colon. *Cholera infantum* is accompanied by serous, *i. e.*, watery stools which contain little mucus or food; the abdomen is flabby rather than distended, and a hydrocephaloid state tends to supervene. (Raue.)

Prognosis.—Intestinal indigestion generally terminates favorably in a few days. Entero-colitis runs a course extending

over two weeks or more, and without careful treatment tends toward a fatal issue. In cholera infantum the outlook is grave, death sometimes occurring in twenty-four hours. In any case a fall in temperature, a lessened frequency of the pulse, and the return of normal constituents to the stool, presages a favorable termination.

Treatment.—In all infantile diarrheas diet is of first importance. During the acute stages withhold food, giving instead barley water, which affords no pabulum for the micro-organisms present in the intestine. Later, allow carefully selected milk, sterilized and diluted with plain lime water. Increase the quantity very gradually by adding a little more to the barley water each day. Insist upon absolute regularity in the hours of feeding.

In the severe forms remove the poison from the gastro-intestinal tract by washing out the stomach, using a soft rubber catheter as a stomach-tube, and irrigating the colon with clear water. General baths may be used to control pain and pyrexia, while a great loss of fluids may demand the use of saline solution, a pint or more in twenty-four hours by hypodermoclysis.

In many cases alcoholic stimulation is advisable; add half an ounce of brandy, the white of an egg, and a little sugar to a tumblerful of water, strain, and administer a teaspoonful every hour (Raue).

In mild cases early febrile symptoms will suggest the use of *aconite* or *belladonna*, while in the absence of fever *nux vomica* is generally indicated. Later manifestations will be met by such remedies as *ipecac* (nausea, vomiting, green stools), *veratrum viride* (purging predominating), *cuprum ars.* (cramps, diarrhea), *chamomilla* (colic, flatulence, fretfulness), and *podophyllum* (thin, copious, painless stools).

In severe cases *arsenic* is demanded by choleraic symptoms, such as severe vomiting and purging, thirst, restlessness, and a tendency to collapse. *Croton tig.* may be suggested by yellow watery stools expelled with great force. *Cuprum ars.*

is valuable in the condition ascribed to arsenic, but with severe nervous symptoms and pain. *Veratrum alb.* is required in conditions of impending collapse, the patient being bathed in cold sweat. Complete collapse calls for the use of *camphor*, *zincum met.* or *cyanide*, or *hydrocyanic acid.*

An excess of mucus in the stools suggests the use of *mercurius*, *sulphur*, or *gummi gutti.*

INTESTINAL OBSTRUCTION.

(Ileus.)

Etiology.—Occlusion of the intestinal canal, partial or complete, may result from a variety of lesions, including the following:

1. *Strangulation.* The intestine may become constricted by inflammatory bands or adhesions, the result of peritonitis; or by the forcing of the bowel into pouches, openings, or omental or mesenteric slits.

2. *Volvulus.* Twists or knots of the bowel are met with principally in the large intestine, especially about the sigmoid flexure of the colon. Strangulation and volvulus are apt to occur in adult males.

3. *Intussusception* or *invagination.* One portion of the bowel may slip down into another from above ("telescope"); this is particularly common in young children as the result of diarrhea or constipation.

4. *Abnormal contents of the bowel.* Foreign bodies, such as gall-stones, enteroliths, or seeds, may occlude the intestine; or fecal impaction, resulting from constipation or from paralysis of a bowel segment, may produce the same result.

5. *Strictures* and *tumors.* Stricture of the bowel may be congenital (imperforate anus) or due to the cicatrices of healed ulcers (tuberculous, syphilitic, dysenteric, etc.). It may be produced by benign or malignant neoplasms.

Symptoms.—Acute Obstruction of the intestine is accompanied by:

1. *Abdominal pain*, sudden and severe.

2. *Vomiting*. This is apt to be copious, persistent, and projectile in character. The vomitus consists first of food, then of bile, and finally of fecal matter. This projectile, stercoraceous vomiting is pathognomonic of intestinal obstruction.

3. *Constipation*. As a rule constipation is absolute from the beginning. In a few cases it becomes so only after the contents of the intestine below the lesion have been discharged.

4. *Distention* is usually discovered in the bowel above the point of obstruction, and it is frequently accompanied by visible peristaltic movements of the intestinal coils in the same locality. Tenderness is usually diffuse, but it may sometimes be found localized to the area about the lesion, and exceptionally a tumor or stricture may be palpated. Tenesmus is characteristic of intussusception, and may be accompanied by the passage of mucoid and bloody stools.

5. *Collapse* usually develops early in the attack. The constitutional symptoms are profound, but no fever is present until peritonitis is set up.

Chronic Obstruction of the intestines is generally due to fecal impaction, foreign bodies, cicatricial strictures, or malignant neoplasms. *Fecal impaction* may be diagnosed upon the discovery of a rectum filled with hard fecal matter, in addition to which a tumor appears sooner or later in the region of the cecum. The impaction may be accompanied by attacks of diarrhea, the intestinal secretions channelling their way through the mass. In cases of chronic obstruction due to other causes the symptoms are not characteristic. As a rule, there is a history of long-standing constipation, paroxysmal abdominal pain, sometimes grooved or flattened stools, and gradual failure in health. Examination usually reveals abdominal distention, and frequently the peristaltic movements of the intestinal coils can be seen through the abdominal wall.

Diagnosis.—In an acute case the coincidence of sudden severe pain in the abdomen, persistent and later stercoraceous vomiting, constipation, distention, and collapsic symptoms renders the diagnosis of intestinal obstruction easy. It is not enough, however, to recognize the nature of the lesion; it is essential that its character and situation shall, if possible, be determined. For this purpose the history, the symptoms, and the signs discoverable on physical examination, both external and per rectum and vagina, are often necessary.

Obstruction to the small intestine is usually indicated by the sudden onset of intense pain; vomiting is early, copious, and persistent; the constipation can be apparently relieved by enemata; collapse is common; distention is not marked; and indican may be found in the urine.

Obstruction to the large intestine is less acute in its onset, the pain is less severe, distention and constipation are early and marked symptoms, and vomiting is delayed and frequently scanty. Collapse is a late symptom. Indican is not found in the urine.

Acute general peritonitis may be distinguished by a history of one of its causal conditions (appendicitis, pelvic infections), the presence of fever, the characteristic pulse, the more uniform distention of the abdomen, the absence of gurgles in the bowel, and the non-projectile character of the vomiting.

Acute enteritis presents, as a rule, diarrhea and fever, without marked distention of the abdomen or stercoraceous vomiting.

Intestinal colic may be excluded by its history, the character and localization of the pain, and the absence of obstinate constipation, stercoraceous vomiting, collapse, and local tenderness.

Prognosis.—In the acute form the outlook is extremely unfavorable, the disease terminating in death within a week if surgery does not intervene. The chronic cases due to fecal impaction may terminate favorably through discharge of the obstructive mass. Chronic cases due to other causes may be

prolonged for weeks, with progressive emaciation and anemia, until finally acute obstruction intervenes and death rapidly ensues. Surgical measures serve to prolong life in many instances.

Treatment. — In cases of acute intestinal obstruction the patient should be placed at rest, food withheld, and morphia, gr. ¼, with atropine, gr. $\frac{1}{120}$, should be given to control the excessive peristalsis. When relaxation has thus been secured, the colon may be irrigated with large quantities of warm water, in an effort to overcome the obstruction. In some cases the patient may be anesthetized and placed in the knee-chest position, in order to facilitate the irrigation. Give no purgatives. If it can be determined that the obstruction is due to fecal impaction an enema of oil may be used, as suggested for the treatment of constipation.

Should relief not be secured within twenty-four hours, immediate operative interference should be urged. In cases of chronic obstruction the treatment should be conducted according to the symptoms present and their supposed cause; but if the obstruction becomes complete and acute symptoms arise, the case must be treated as though it had been acute from the start.

APPENDICITIS.

Etiology.—The principal cause of appendicitis is the anatomical structure of the vermiform appendix itself. This useless, feebly-nourished tube, which receives its blood supply from a single artery, affords ready ingress to irritating matters, and expulsion of the latter is difficult. In women, in whom a branch of the ovarian artery affords a more perfect blood supply, appendicitis occurs much less frequently than in men. Youth, the male sex, intestinal indigestion, exertion (probably by causing twists of the appendix), and the presence of foreign bodies may predispose to the attack; but the exciting cause is invariably bacterial in nature (bacilli coli, streptococci, staphlyococci, etc.).

Pathology.—Inflammations of the appendix may differ in their intensity and in the extent of tissue-involvement; and accordingly the following may be recognized:

1. *Endo-appendicitis.* The mucous membrane lining the appendix undergoes catarrhal or purulent inflammation; and this may result in constriction of the outlet and the formation of a retention-cyst, or, occasionally, in adhesion of the walls and obliteration of the sac (*appendicitis obliterans*). Frequently, however, this stage is followed by:

2. *Parietal* or *interstitial appendicitis.* The ulcerative process extends, involving all the coats; the swelling constricts the tube, and minute abscesses are formed, or gangrene, either localized or diffuse, supervenes. This stage invariably terminates in necrosis or gangrene, with perforation or rupture into the peritoneal cavity.

3. *Peri-appendicitis* and *extra-appendicular lesions.* By its extension the inflammation leads to:

(*a*) *Circumscribed peritonitis*, either with adhesions between the appendix and the adjacent tissues, or the formation of a localized abscess. According to the position of the appendix, this abscess may point inwards, its rupture leading to diffuse septic peritonitis; or it may point inwards and downwards, and in that case the pus may rupture into the rectum, bladder, or vagina; or it may point upward along the "lateral gutter," and if not evacuated it may finally penetrate the diaphragm, or the abdominal wall near the umbilicus. Occasionally the abscess is retro-peritoneal ("iliac abscess"), and may, finally, penetrate in the neighborhood of Poupart's ligament. The ileum or the cecum may be involved by the direct spread of the appendical inflammation; and occasionally the liver is invaded by a thrombus or by septic emboli.

Diffuse septic peritonitis may directly follow perforation, or it may be delayed until the adhesions limiting the latter have given way.

Symptoms.—As a rule the onset of appendicitis is attended by a majority of the following symptoms:

1. *Sudden pain.* which may be felt anywhere in the abdomen but tends to localize itself in the right iliac fossa.

2. *Tenderness* in the right iliac fossa, especially at McBurney's point, two inches from the anterior superior spine of the right ilium on a line drawn from that point to the umbilicus.

3. *Rigidity* of the right rectus muscle.

4. Moderate *fever* and *rapid pulse.*

5. *Gastro-intestinal disturbances.* The tongue is coated, and vomiting is not unusual; constipation is generally present, but diarrhea is not uncommon.

6. Early there is a tympanitic *tumor*, reducible by pressure; later, if an abscess develops, there will be a hard tumor, also tympanitic.

Varieties.—Appendicitis may be acute, hyperacute, or subacute in its onset; it may be primary, or may supervene upon another disease (typhoid, tubal disease); and it may be chronic, either *recurring* after long intervals of apparent recovery during which no tenderness is present, or *relapsing* into a new attack before the previous one has fairly disappeared.

Course.—Should an attack of appendicitis tend to recovery, the pain and rigidity quickly lessen, and in the course of a few hours the temperature and pulse-rate fall to normal. Should suppuration be present, the temperature usually remains high; but either suppuration or gangrene may be unattended with fever.

Should rupture into the peritoneal cavity ensue, it may be indicated by a sensation of something having broken in the abdomen, followed by severe pain; and the resulting *general peritonitis* will give rise to the following symptoms:

1. Severe diffuse, not localized, pain.

2. Uniform distention and tenderness of the abdomen, with absence of gurgle.

3. Moderate fever; sometimes a subnormal temperature.

4. A rapid, feeble pulse; and a dry, coated tongue.

5. Collapsic symptoms: pinched face, hollow eyes, cold, clammy skin, feeble pulse, and death.

Diagnosis.—In typical cases the recognition of appendicitis is easy, particularly if the cardinal symptoms of pain, tenderness, and rigidity are kept in mind; but at times a number of other conditions may closely simulate the disease.

Acute intestinal indigestion frequently resembles appendicitis and in fact may usher in the latter disease. The pain of indigestion is usually higher in the abdomen, and diarrhea is more common than in appendicitis; but the classical symptoms of the latter may develop at any time.

Acute intestinal obstruction presents no fever, localized pain and tenderness are less marked, and vomiting is persistent and stercoraceous in character. Visible peristaltic movements may be observed above the point of obstruction.

Renal colic is accompanied by pain which extends down the ureter and along the spermatic cord, localized tenderness is generally absent, and blood or small concretions may be discovered in the urine.

Biliary colic is accompanied by pain in the region of the gall-bladder and extending toward the umbilicus, tenderness is often found over the Mayo-Robson point, and jaundice frequently appears.

Perinephritic abscess may simulate appendicitis, but as a rule no intestinal symptoms are present, and the history is suggestive of chronic renal disease or nephrolithiasis.

Malignant Disease.—A cancerous mass in the neighborhood of the cæcum may produce symptoms of obstruction and pain strongly suggestive of chronic appendicitis, and frequently operation alone can settle the question.

Floating kidney, by presenting vague pain and a tumor in the right side of the abdomen, may resemble chronic appendicitis; but as a rule the history and physical examination will disclose the real nature of the condition. A floating kidney predisposes the patient to appendicitis.

Pelvic affections in women are, as a rule, more gradual in

their onset and may be recognized by skillful examination *per vaginam.*

Extra-uterine pregnancy may be distinguished by the menstrual history, the lack of fever, and the collapsic character of the symptoms which follow rupture; and examination may reveal a tumor in the pelvis.

Typhoid fever is gradual in its onset, and presents a peculiar temperature curve and a characteristic eruption at the end of a week. Rigidity of the abdominal wall is not prominent in typhoid, but in cases attended with early perforation the diagnosis may be very difficult, for it must be remembered that appendicitis sometimes develops in connection with typhoid fever.

Prognosis.—While it cannot be doubted that the majority of cases of appendicitis will recover without operation, in no case can the symptoms be trusted to afford an exact indication as to the outcome. Moreover, recurrence is extremely common. It is, therefore, difficult to frame a prognosis based solely upon the probable results of medical treatment.

The position and direction of the tenderness afford indications of varying gravity, which may be utilized in framing a prognosis and determining to what extent treatment may venture to be expectant. If the appendix points inward or inward and upward, it is very dangerous; if inward and downward into the pelvis, as is usual in women, it is less dangerous; if outward and upward in the lateral gutter, it is very safe (Van Lennep.)

Should suppuration occur, surgical intervention alone can be relied upon to save the patient, although in rare instances rupture of the pus into bowel, bladder, or groin has been followed by recovery. General peritonitis renders the prognosis almost hopeless.

Treatment.—As soon as appendicitis is suspected a surgeon should be called in consultation and as a rule operation advised. Pending this, or when operation is refused, the patient should be placed at rest in bed with an ice-bag over the

right iliac region, and the large bowel unloaded by means of a high rectal enema. If it is certain that suppuration has not occurred and the disease is subsiding, a saline purge may be given. Opiates must be used only after all questions as to diagnosis and treatment have been decided. Liquid foods alone are allowable. Early in the attack such remedies as *belladonna*, *bryonia*, *colocynth*, *cuprum*, *nux vomica* and *sulphur* may be given; the onset of suppuration calls for *mercurius* or *hepar;* and peritonitis frequently affords indications for the use of such medicines as *arsenic*, *cantharis*, and *mercur. corr.*

CONSTIPATION.

Etiology.—Constipation may be due to many causes, the more important of which are:

1. Habit, due to a persistent disregard of the natural inclination to defecation.

2. Atony of the bowel, with lessened peristalsis; this may be due to local disease or general debilitating affections (fevers, chlorosis, etc.).

3. Deficiency of the secretions which normally stimulate peristalsis, *e. g.*, bile.

4. Improper food. Vegetable foods which have a solid residue, such as cereals, stimulate peristalsis; but milk, leaving no residue, has a contrary effect.

5. Atony of the abdominal walls (over-distention, obesity).

6. Stricture, due to impingement on the bowel of displaced organs (*e. g.*, uterus), tumors, or foreign bodies.

Treatment.—1. Ascertain and if possible remove the cause.

2. Cultivate a regular habit, going to the closet at a certain hour each day whether inclined or not.

3. Adopt a suitable diet; fresh vegetables, fruits, bran or gluten bread, and the free use of water between meals.

4. Massage the abdomen daily. Flex the body repeatedly while in the standing position.

5. Enemata may be used in obstinate cases. Plain water or soap and water may give temporary relief, especially if injected into the colon. In very protracted cases with fecal accumulation, place the patient in the knee-chest position and inject slowly eight or more ounces of olive or cotton-seed oil. This should be done by the patient or his attendant at his own home and preferably at bedtime; for the patient must retain the knee-chest position for twelve minutes, then turn gently on to his right side and lie for some hours without turning over. This procedure is followed in many instances by a thorough emptying of the large bowel and complete relief of the disorder.

6. Medicines. Never use a purgative if it can be avoided. More useful medicines are *nux vomica*, suggested by ineffectual urging to stool, with headache, nausea, bad taste in the morning, and often hemorrhoids, and *strychnine*, which in atonic cases may be substituted for nux. Cases with symptoms of portal hyperemia, especially if diarrhea alternates with constipation, are benefited by *sulphur*. *Hydrastis* is suggested by sensations of epigastric weakness and sinking, sour eructations, hemorrhoids, and hard stools coated with mucus. *Opium* is indicated for the complete intestinal torpor which occurs in old people or which follows acute diseases or lead colic; the feces are hard and dry. *Bryonia* may be given when similar stools are due to a lack of intestinal secretion, especially in irritable hypochondriacs. Many other remedies may be suggested by associated symptoms.

INTESTINAL CANCER

Intestinal cancer (usually epithelioma, less often colloid or scirrhus) is rare. When it does occur it is usually primary and in a great majority of cases attacks the rectum, where it is easily recognizable. It may be annular or diffusely nodular. Tumors occurring high up in the intestine present no characteristic symptoms; but evidences of chronic obstruction of the bowel together with persistent pain and

constipation may suggest the nature of the disease, and if in addition there are cachexia and altered stools (pus, blood, fragments of cancerous tissue) a definite diagnosis is possible. Physical examination may reveal a sensitive tumor which does not indent on pressure, and which gives a dull tympanitic percussion note; if it is in the colon or small intestine it is freely movable. The prognosis is absolutely unfavorable, although surgical measures may prolong life in some cases.

INTESTINAL NEUROSES.

1. Secretory.

Membranous enteritis is a rare disease characterized by the discharge of a membrane-like substance with the stools. It occurs in neurotic individuals, usually women past forty. The course is chronic, and treatment should be directed to the underlying hysteria or neurasthenia.

2. Sensory.

Enteralgia (neuralgia of the bowel) consists of attacks of intestinal pain unaccompanied by organic disease of the bowel. It may be neurotic or gouty in origin, it may be due to irritation of the intestine by its contents, sometimes it is reflex from diseases of the other viscera, and occasionally it is due to "central causes," *i. e.*, diseases of the brain or spinal cord, such as locomotor ataxia or spinal caries. Metallic poisons, especially lead, cause severe intestinal pain. The diagnosis and treatment should be directed toward the cause, but temporary relief may often be secured by the use of such symptomatic remedies as *colocynth*, *nux vomica*, *belladonna*, *cuprum arsen.*, *plumbum*, and *dioscorea*.

3. Motor.

Nervous diarrhea may occur as the result of central nervous disturbances (*e. g.*, in locomotor ataxia), and in neurotic individuals it may be due to emotional disturbances, such as joy or fright. A simple increase in the number of stools, without change in their consistency, is the usual symptom.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Etiology.—Inflammation of the peritoneum is rarely, if ever, primary; as a rule it is secondary to:

1. Inflammations of the gastro-intestinal tract (perforating appendicitis, gastric or intestinal ulcer, toxic gastritis, intestinal obstruction, strangulated hernia, malignant tumors).

2. Pyogenic inflammation of the pelvic viscera (puerperal, gonorrheal, etc.).

3. Traumatism (wounds, blows, operative incisions).

4. Certain general diseases, such as rheumatism and nephritis.

Pathology.—As the result of bacterial infection the peritoneum becomes hyperemic and exudation ensues; the latter may be:

1. *Fibrinous.* The exudate is composed of flakes of yellow lymph which tend to glue together the intestinal coils.

2. *Sero-fibrinous.* The fibrin produces adhesions and the serum gravitates to the flanks.

3. *Purulent.* Pus is present in varying, often large, amounts, and the inflammation tends to become gangrenous.

4. *Hemorrhagic* effusion may occur, especially in cancerous and tuberculous cases.

In extent peritonitis may be:

a. Circumscribed, the inflammatory process being localized by adhesions and virtually constituting an abscess. The latter may, however, rupture into the general peritoneal cavity and set up diffuse peritonitis.

b. General or *diffuse*, the peritoneal surface of the intestinal coils being more or less completely covered with a yellowish exudate.

Symptoms.—The onset of acute general peritonitis is sudden, and is accompanied by:

1. *Pain*, which is often extremely severe and is rendered agonizing by the slightest movement. Its location usually corresponds to the area of inflammation.

2. *Tenderness*, which is similarly localized. It is so intense that in order to relieve the tension of the abdominal walls the patient lies on his back with the knees drawn up.

3. *Abdominal distention*, due to paralysis of the muscular coat of the bowel. This paretic condition leads to constipation.

4. *Vomiting*, at first of the stomach contents and later of bile.

5. *A small, rapid, weak pulse.* This is characteristic, while pyrexia, though usually present, is variable in degree.

6. *Collapse*, with cold, clammy skin, pinched face and sunken eyes (Hippocratic expression) together with complete consciousness and intense mental anxiety.

Physical Signs.—In addition to abdominal distention and tenderness, the patient may survive long enough for signs of effusion to appear, *i. e.*, percussion-dulness and fluctuation discoverable first in the flanks and ascending as the amount of fluid increases. The gurgling sounds normally heard in the intestines are absent and the belly is silent, indicating complete intestinal paralysis.

Acute Circumscribed Peritonitis, while presenting these symptoms in milder degree, remains localized; and in addition to the pain and tenderness, the fluctuation and percussion-dulness of an abscess may be recognized. The latter tends, as stated, to ultimately rupture into the general peritoneal cavity, with fatal results, but it may instead penetrate the stomach, bowel, or abdominal wall.

Diagnosis.—*Acute enteritis* is accompanied by pain and distention, but the pain is colicky, may intermit, is accompanied by diarrhea, and severe constitutional symptoms are absent.

Intestinal obstruction is accompanied by projectile fecal vomiting, and lacks extreme tenderness, wiry pulse, and pyrexia (until peritonitis supervenes).

Hysterical abdomen may resemble peritonitis, but the associated history and conditions, the absence of fever and constitutional disturbance, and the disappearance of the alleged tenderness when the attention is distracted, will usually serve to enlighten the attendant.

In *enteralgia* the pain is relieved by pressure, fever is absent, and the absence of associated conditions serve to render diagnostic confusion improbable.

Prognosis.—General peritonitis is almost invariably fatal within a week. Localized peritonitis may recover through surgical intervention, or, rarely, spontaneous discharge outside the peritoneal cavity.

Treatment.—Abdominal section offers slight hope; but as a rule the use of morphine hypodermatically to control pain, ice or champagne to relieve vomiting, and liquid food to conserve the strength, constitute the main features of the treatment of hopeless septic cases. When the peritonitis is circumscribed, local applications of cold or heat may aid in checking its advance, but surgical interference early in the attack is advisable.

In localized peritonitis the early symptoms may be combated with *aconite* or *veratrum viride*, or, in young children, *belladonna*. Upon the development of the characteristic pain, *bryonia* is indicated. Advanced cases with impending heart failure may be given *arsenic*. Intensely septic cases afford indications for *arsenic*, *cantharis*, *mercurius corr.*, or the *snake poisons*. Typhoid symptoms may suggest the use of *rhus tox*. Failing circulation demands the use of alcoholics or of *camphorated oil* or *strychnine* hypodermatically.

Chronic Peritonitis is usually tuberculous, but it may be due to cancer, syphilis (in infants), or inflammation of one of the abdominal organs involving its peritoneal covering; or it may remain as a sequel of an acute attack. The peritoneum may become thickened with connective tissue, the intestines may be matted together with adhesions, or there may be a varying amount of effusion present. Symptoms may result from in-

testinal obstructon produced by the adhesions, or there may be more or less vague pain; but in many cases no subjective evidence of the disease is present. Discovery of the presence of fluid in the abdominal cavity may lead to a diagnosis of the condition. The treatment should include the administration of such remedies as *apis*, *aurum*, *kali iod.*, and *sulphur;* and in many cases repeated paracentesis is necessary.

ASCITES.

Etiology.—Abdominal dropsy may result from:

1. Portal obstruction, either within the liver (cirrhosis) or without the liver (thrombosis, or pressure by neoplasms, aneurism, peritonitis, etc.).

2. General venous stasis, due to chronic heart or lung disease.

3. Cachectic states associated with hydremia (anemia and nephritis).

Symptoms.—By its mechanical effect the serous accumulation produces abdominal distention with uneasiness and discomfort, while pressure upon the veins may cause secondary edema of the lower extremities. The latter will, however, precede the abdominal accumulation when the dropsy is due to general venous stasis. The ascitic fluid is freely movable, and with each change of the patient's position it gravitates to dependent parts. The abdominal viscera may become displaced and the diaphragm crowded upward, as a result of which respiration may be seriously embarrassed.

Physical Signs.—When the patient stands erect the lower portion of the abdomen bulges forward; when he lies down it flattens, and the flanks bulge. Placing the hand on one side of the abdomen and tapping with the fingers on the opposite side, a succussion wave will be felt. By percussion absolute dulness is elicited over the fluid, and this dulness alters its location with each change of the patient's position.

Diagnosis.—An *ovarian cyst* may resemble ascites; but the

cyst is unilateral at first, rises up toward the centre of the abdomen and does not distend the flanks, and the umbilicus does not become protuberant as in ascites. Vaginal examination may aid the diagnosis. If necessary a small portion of the fluid may be withdrawn by the needle; if ascitic, it is generally clear and straw colored, with a specific gravity of 1010 to 1014; if ovarian, it is turbid, of varying color, and its specific gravity is 1020 or more.

Chronic peritonitis with effusion is generally tuberculous. It presents some pain and pyrexia, and is usually associated with manifestations of tuberculosis elsewhere. If adhesions are present the fluid changes its position less readily than in ascites, and on withdrawal it is found to be more albuminous and of higher specific gravity than ascitic fluid.

A *distended bladder* has been mistaken for ascites, but its origin is low in the pelvis and it disappears on catheterization.

Treatment.—1. Discover and treat the causal condition.

2. In severe cases the use of such remedies as *apis*, *arsenic*, *cinchona*, *helleborus*, or *iodine* is required; or, if these fail, administer diuretics, such as infusion of *apocynum* or of *digitalis*, 20 drops every 3 or 4 hours.

3. Paracentesis should be used only when other measures have failed and the condition is extreme. Puncture with a straight trocar at a point midway between the symphysis and umbilicus, and withdraw the fluid slowly. Use strict antiseptic precautions.

DISEASES OF THE LIVER AND GALL-DUCTS.

Diseases of the liver may produce many and varied symptoms, both local and general; but as a rule attention is particularly attracted to that organ by the occurrence of local pain, jaundice, ascites, or dyspeptic symptoms.

1. *Hepatic pain* may be felt in the region of the liver, or in the right shoulder tip. More often the sensation is one of vague discomfort, or a feeling of distention in the right hypo-

chondrium calls attention to that region. Intense agonizing pain is felt when the bile duct has become occluded, *e. g.*, by a stone (*biliary colic*).

2. *Jaundice* is invariably the result of some disease of the liver or the gall-passages. The skin, conjunctivæ, and mucous membranes become uniformly pigmented in shades varying from a light yellow to a yellowish green. This icterus is generally first detectable in the conjunctiva or in the mucous membrane under the tongue. It is accompanied by itching of the skin, the urine and saliva are tinged with yellow, and the feces are often white from lack of bile.

3. *Ascites*, the abdominal dropsy which results from obstruction to the return of blood to the heart by way of the portal circulation, is most often due to disease of the liver (cirrhosis, etc.).

4. *Dyspeptic symptoms* may be of hepatic origin, as the result of:

(*a*) The absence of bile from the intestine, in consequence of which the feces are white in color, and decomposition of the intestinal contents takes place, this latter producing flatulence; or

(*b*) The occurrence of venous stasis in the gastro-intestinal circulation, which, by inducing passive congestion and catarrhal changes in the mucous membranes, leads to various symptoms of irritation in the stomach and intestines.

Interrogation of the Patient.—When an affection of the liver is suspected, careful inquiry should be made as to:

1. A *history* of dietetic errors, alcoholism, gastric catarrh, previous attacks of jaundice, companionship with dogs (echinococcus), syphilis, prolonged suppuration (amyloid degeneration), or exposure to poisons, especially phosphorus.

2. *Pain.* What is its location? Has he ever had an attack of severe pain coming on suddenly and lasting for some hours? Did the pain at that time radiate, and if so, in what direction? Was there vomiting with it? Was he yellow afterwards? Does he ever have pain in the tip of his shoulder?

3. *Jaundice.* Has he noticed any change in the color of his skin, his urine, or his feces? Does his skin itch?

4. *Venous stasis.* Is his abdomen swollen? Does he suffer with piles? Has he ever vomited blood? Does he suffer with gastro-intestinal symptoms, such as epigastric discomfort, distention, belching, constipation, etc.?

Examination of the Patient.—Inspection of the hepatic region may reveal nothing of importance. In some cases, however, it may be noticed that the abdominal veins have become dilated and prominent, or even varicose and serpiginous in the neighborhood of the umbilicus (*caput Medusæ*), thus indicating the establishment of collateral avenues to compensate for portal obstruction. A localized edema may be noted (abscess of the liver), the hepatic region may be diffusely elevated (hepatic hypertrophy), or an isolated pyriform swelling may be observed in the middle of the epigastrium (dilated gall-bladder). Rarely the lower margin of the liver can be seen moving up and down with respiration. On inspection we may also discover pulsations in the liver (heart disease).

Palpation.—Place the patient on his back with the abdominal walls relaxed, distract his attention with conversation, and place one hand flatly on the abdomen, just outside of the rectus muscle. Depress the side of the hand slightly so as to push up a fold of skin and ask the patient to take a deep breath; as he does so the margin of the liver may be felt to ride over the edge of the hand. If this method is unsuccessful, place the left hand against the right lumbar region and endeavor to press the liver upward, while the right hand makes gentle continuous pressure and gradually works down so as to reach the border of the liver. If the examiner is in doubt as to whether he really feels the edge of the liver, let him search for the fissure of the gall-bladder, or for that near the median line produced by the round ligament.

In health the liver is rarely palpable, but when increased in size, as the result of hyperemia, obstruction in the gall-ducts, inflammation, syphilis, malignant disease, fatty infiltration, or

amyloid degeneration, the organ may extend several finger-breadths below the border of the ribs. The surface of the liver may be felt to be granular (cirrhosis), or nodular (carcinoma), and its edge may be smooth and rounded (amyloid disease). In cases of enlargement due to circulatory, inflammatory, or degenerative processes which involve the organ as a whole, the normal shape is usually preserved, and this may serve to exclude such conditions as cancer, abscess, or hydatid cyst. The liver, and consequently tumors attached to it, move with respiration.

By palpation the degree of *pain* can be ascertained. Pain accompanies disease of the liver only when its peritoneal covering is inflamed (perihepatitis, syphilis, cancer) or rapidly distended (hyperemia, abscess, hydatid cyst). Pain is also the result of irritation of the gall bladder or ducts. *Perihepatic friction* is sometimes felt over the liver. It consists of a peculiar sensation like the creaking of leather, communicated to the examiner's hand and also heard during auscultation; it is produced by the rubbing together of the roughened hepatic and parietal layers of the peritoneum. The *hydatid thrill* is a sensation like that felt on percussing a bladder filled with water. To elicit it, place the left hand on the tumor and note the vibration produced by percussing in its neighborhood with the right hand. The hydatid thrill is not invariably present in echinococcus cysts, and it is also met in connection with ovarian cysts and even simple ascites. Palpation may also reveal morbid conditions of the gall bladder; the latter cannot be detected unless distended, when it may be felt as a smooth, pear-shaped tumor, extending out toward the umbilicus. It may be enlarged, soft, and fluctuating (dropsy), or it may contain gall-stones and impart a sensation like that felt on squeezing a bag of marbles.

Percussion.—In order to permit percussion of the liver anteriorly, the patient should lie on his back, but for percussion posteriorly he must stand, sit, or lie on his left side. The normal limits of dulness are as follows:

		Median Line.	*Mammary Line.*	*Mid-axillary Line.*	*Scapular Line.*
Upper border.	Deep percussion-dulness (use forcible percussion).	Blends with heart dulness.	4th interspace.	7th interspace.	9th interspace.
	Superficial percussion-dulness.		6th rib.	8th rib.	10th rib.
Lower border (use light percussion and pass upwards).		A hand's breadth below the base of the xiphoid.	About the costal margin.	10th interspace.	Blends with kidney dulness.

MALFORMATIONS AND MALPOSITIONS OF THE LIVER.

Malformations.—Alterations in the shape of the liver may be congenital or acquired. Syphilis may produce a lobulated liver which pathologically presents the changes of a chronic interstitial hepatitis; and the symptoms are those of the latter disease. The "corset liver," in which a furrow across the anterior and lateral surface is produced by compression, is very common in women, but occasions no direct symptoms. In some of these cases careful palpation along the edge of the liver may be necessary in order to distinguish the lower portion from an abdominal tumor.

Malpositions.—The liver may be transposed, appearing mainly on the left side. This is usually associated with transposition of the other viscera. It may be displaced in various directions, as the result of tight lacing, or by the pressure of pleuritic effusion, emphysema, ascites or abdominal tumors. The "*wandering liver*," which is permitted by an abnormally long suspensory ligament to fall out of its usual position, generally occurs in middle-aged women with lax abdominal walls (*splanchnoptosis*). The organ may be recognized as a large, hard, movable tumor, corresponding in shape and size to the liver, while the normal site of the latter is tympanitic on percussion. Various dragging sensations and nervous symptoms may be induced by the displacement. The treatment is limited to the application of an abdominal supporter.

CIRCULATORY AFFECTIONS OF THE LIVER.

Anemia of the liver occurs in connection with a general anemia, or with fatty or amyloid degeneration of the organ, but it occasions no symptoms and is discovered only post mortem.

Active Hyperemia (congestion) of the liver takes place physiologically after each meal, and it may be exaggerated by indulgence in rich food or alcohol, and may even eventuate in interstitial changes. It also accompanies malaria and other infectious diseases, and suppression of menstruation. It causes no characteristic symptoms.

PASSIVE HYPEREMIA OF THE LIVER.

Etiology.—Passive hyperemia of the liver results from obstruction of the flow of blood from the liver. Its cause may be:

1. Local, such as pressure over the portal area from without (by a tumor or cyst), venous thrombosis, etc.

2. General, especially in connection with the venous stasis of uncompensated heart disease, less often as the result of chronic pleurisy, emphysema, and intra-thoracic tumors.

Pathology.—The central vein of each lobule and its adjacent capillaries become congested, and by contrast with the paler periphery of the lobule, an alternation of tints is produced which is recognized post mortem as the "*nutmeg liver.*" Later, atrophy is induced, the central cells and capillaries being replaced with dark pigment and connective tissue (*red* or *cyanotic atrophy*).

Symptoms.—The liver becomes enlarged and tender, and its lower edge can be detected considerably below the border of the ribs. The patient complains of discomfort in the right hypochondrium, together with such gastro-intestinal symptoms as a furred tongue, nausea, vomiting, diarrhea, or constipation. There may be slight jaundice or ascites, or general anasarca may be present. Congestion of the gastric mucous

membrane, hyperemia and enlargement of the spleen, and congestion of the kidneys, in consequence of which the urine is scanty and of high specific gravity, are more or less constantly associated with hyperemia of the liver as results of the same general condition, *i. e.*, venous stasis.

Treatment.—This must be directed to the causal condition, most frequently heart disease with broken compensation. In many of these cases *digitalis* and other heart tonics fail to act until the dropsy and portal stasis have been removed, and the latter may be accomplished by the administration of *mercurius dulcis*, 1x, in doses of one grain every two or three hours for several days.

PERIHEPATITIS.

Etiology.—Inflammation of the peritoneal covering of the liver may result from:

1. The spread by continuity of a hepatic inflammation (abscess, hydatid cyst).
2. Perforation of the stomach or intestine (ulcer, appendicitis, etc.).
3. The spread of pleurisy through the diaphragm.
4. Direct violence, such as a blow, or continued pressure.
5. General peritonitis.

Pathology.—The inflammation may be plastic, a fibrinous exudate leading to the formation of adhesions, but more often it is purulent, the space between the liver and the diaphragm becoming filled with circumscribed areas of pus (*subdiaphragmatic* or *subphrenic abscess*). Air or gas derived from the lung or gastro-intestinal tract may also be present (*subphrenic pyopneumothorax*). In a more chronic form of perihepatic inflammation the capsule becomes thickened and attached by adhesions to adjacent organs, and ultimately the liver itself becomes shrunken and its vessels and ducts occluded.

Symptoms.—In acute cases the onset is attended with the circumscribed pain and tenderness characteristic of a localized

peritonitis, while rigors and irregular pyrexia may announce the formation of an abscess. During the early stage a friction rub may be heard over the hepatic area, and when an abscess has formed the right hypochondrium becomes distended, the intercostal spaces appear motionless, the line of percussion dulness is elevated, and the lower border of the liver is depressed.

Course.—The duration is extremely variable; if the exudate is fibrinous, resolution may occur in a few days; but if the process is purulent the disease may be prolonged, death finally resulting from asthenia or rupture of the abscess into a neighboring organ. Rarely the pus is absorbed or is evacuated through the abdominal wall, and recovery follows.

Diagnosis.—*Empyema* of the right pleural cavity may be distinguished by the presence of cough and expectoration, and the displacement of the heart to the left. If there be doubt, aspirate in the seventh or eighth interspace in the midaxillary line; the presence of bile pigment in the pus, sometimes sufficient in amount to render the latter very yellow, indicates the subdiaphragmatic location of the abscess.

Prognosis.—The fibrinous variety terminates favorably in a short time. In the absence of surgical interference the purulent cases generally prove fatal.

Treatment.—In an acute case it is essential that the patient be placed at rest and his suffering controlled by hot fomentations and opiates if necessary. *Aconite* may be given at the outset, but as the local inflammation develops, *bryonia* or *asclepias* is usually indicated. *Rhus tox.* may be given in cases resulting from traumatism, and *sulphur* in those whose course is protracted. *Hepar*, *mercurius*, or *silica* may be administered when suppuration occurs, but at the same time appropriate surgical measures become necessary.

ABSCESS OF THE LIVER.

(Suppurative Hepatitis.)

Etiology.—Suppuration within the liver is due to the presence of pyogenic micro-organisms, the sequence of:

1. Traumatism, either by external injury, or by foreign bodies, or parasites (echinoccoci).

2. Infection through the portal vein. This may follow intestinal ulceration (especially dysentery), rectal operations, abdominal abscess, or infectious diseases of the portal vein itself (*suppurative pylephlebitis*).

3. Infection through the hepatic artery, (pyemia, septic emboli).

4. Infection through the umbilical vein, due to infection of the navel in the new-born.

5. Infection through the bile-duct, a sequel of cholangitis.

6. Infection from adherent adjacent organs, such as an ulcerated or cancerous stomach.

Certain cases in which the infection cannot be traced are classed as idiopathic. Adult males are most frequently attacked.

Pathology.—The abscess may be single, as in the tropical (amœbic) variety, or multiple, as in pyemic cases. In a majority of cases the right lobe of the liver is affected. The abscess may penetrate the diaphragm and lung, it may burrow into the peritoneal cavity, into the pericardium, into the adjacent abdominal viscera, or through the abdominal wall externally. If rupture does not occur and life is preserved, encapsulation may take place.

Symptoms.—The abscess may be unsuspected, the symptoms resembling those of typhoid (high fever, delirium, tympanites, enlarged spleen, and even a roseolous eruption), of malaria, (quotidian intermittent fever), or tuberculosis (cough, dyspnea, emaciation, anemia, night sweats, etc.). As a rule, however, tenderness and pain may be discovered in the hepatic region, and often pain is felt in the shoulder-tip. The fever is septic in type, often high (104–105°), and accompanied by chills and sweats. Jaundice is not usual. The liver may be enlarged upward, and as suppuration progresses, fluctuation may be detected.

Diagnosis.—An abscess may be overlooked for a time, though the possibility of its occurrence should be suggested by a history of dysentery, gall-stone colic, injury about the liver, or residence in the tropics. *Tuberculosis* may be excluded by examination of the lungs and sputum; *malaria*, by examination of the blood, which presents leucocytosis and no plasmodium; and *pleural effusion*, by the diminished fremitus and resonance which are found over the area of dulness. *Hepatic intermittent fever*, due to an impacted calculus, offers a history of biliary colic, together with more marked jaundice and less grave general symptoms. A *hydatid cyst* may require aspiration for the discovery of its character. In doubtful cases the patient should be anesthetized and the exploring needle used, with antiseptic precautions.

Prognosis.—This is generally unfavorable, although evacuation of the abscess and free drainage has greatly reduced the mortality. Multiple pyemic abscesses are invariably fatal.

Treatment.—Support the patient's strength with nourishing liquid food and stimulants. *Baptisia*, *belladonna*, *bryonia*, *hepar*, *lachesis*, *mercury*, or *quinine arsenite* may be indicated in the early stages, but when evidences of suppuration are present the abscess must be opened and drained.

CIRRHOSIS OF THE LIVER.

Etiology.—The term "cirrhosis" comprises a group of diseases which have as a common feature the development of fibrous tissue within the liver. The causes may be:

1. Toxic, *i. e.*, poisons taken as food or drink or as medicinal agents (alcohol, spices, etc.).

2. Infective, the source of the infective agents being local (inflammations of the stomach, intestinal, or bile passages) or general (malaria, scarlatina, etc.).

It is probable that various irritants by setting up a gastritis lead to the formation of toxins in the stomach or intestines. The latter are absorbed into the portal circulation, and carried

to the liver, where they induce chronic inflammatory changes, either by their own direct action, or indirectly by destroying the resistance of the liver to the action of certain micro-organisms.

Syphilis causes a special form of hepatic cirrhosis. The liver may also become involved in the general sclerotic changes of senility; and fibrosis often follows long-continued passive congestion.

Pathology.—The liver may be enlarged (*hypertrophic*) or diminished in size (*atrophic*), the latter frequently representing the terminal stage. The organ is hard and the cut surface appears yellowish, brownish, or mottled. The newly-developed fibrous tissue may form a coarse net-work which permeates the whole organ and encloses in each mesh a number of lobules (*multilobular* or *Laennec's cirrhosis*), or a finer network of connective tissue, in which a plexus of bile ducts is imbedded, may surround single lobules (*unilobular* or *biliary cirrhosis*). In another form the connective tissue penetrates the lobules themselves, surrounding single cells or groups of cells (*intra-lobular* or *peri-cellular cirrhosis*), and in a fourth variety dense bands of cicatricial tissue traverse the liver irregularly and by their contraction produce considerable deformity of the organ (*gummatous* or *syphilitic cirrhosis*). As a result of the pressure induced by these changes the nutrition of the secreting cells of the liver is interfered with, and they undergo progressive degeneration. In the course of time the contraction of the connective tissue leads to obstruction of the portal circulation, and ascites results.

Symptoms.—The disease is gradual in its onset and occasions no characteristic symptoms until obstruction to the portal circulation has induced congestion of the stomach and intestines. The first symptoms, therefore, are *dyspeptic* in character; the patient complains of anorexia, eructations of gas, distention, gastric discomfort, and constipation. As the portal obstruction increases, *ascites* develops, and the dropsical accumulation often becomes enormous. *Hemorrhoids* are usually

present. As the gastro-intestinal symptoms increase in severity, *hemorrhages* may take place from the stomach or bowels, and sometimes from the esophagus or nose. In an attempt to form a collateral circulation, the venous channels of the abdomen dilate and the surface becomes marked with varicose veins, notably around the umbilicus, where their ramifications may produce the so-called *caput Medusæ*. Jaundice is rarely present, but the skin is nearly always sallow, and the patient becomes emaciated. Edema of the legs is a late symptom, due to the pressure of the ascitic accumulation upon the blood returning from the lower extremities. The urine is scanty, high-colored, and loaded with bile pigment and urates. Toward the close of the disease toxemic symptoms, such as drowsiness, coma, or delirium, may supervene.

Hypertrophic Cirrhosis—Enlargement of the liver, while often representing merely an early stage of cirrhosis, may in some cases be accompanied by no tendency to contraction. Such cases are distinguished by early and deepening jaundice, by absence of gastro-intestinal irritation and of ascites and other symptoms of portal obstruction, and by the presence of tenderness over the liver. The urine is bile-stained and the urea is normal in amount. The course of the disease is often rapid, terminating with toxemic symptoms (coma or delirium) within a year or two.

Diagnosis.—In the early stages diagnosis is very difficult. The disease often remains unsuspected until ascites or gastro-intestinal hemorrhage leads to examination of the liver. If in the absence of acute disease or starvation that organ is found to be reduced in size, and especially if the spleen is enlarged, cirrhosis may be inferred. It is essential that the liver be examined after the ascitic accumulation has been removed and meteorism reduced, for the presence of either of these conditions causes an apparent diminution in the size of the organ.

Chronic peritonitis, with its liquid effusion, may be mistaken for the abdominal dropsy of cirrhosis, but in the former there is abdominal tenderness, the liver is not diminished in

size, and the fluid obtained by paracentesis is apt to be turbid and to contain matters suegestive of its inflammatory origin.

Hypertrophic cirrhosis may be confused with *cancer* of the liver, but the latter is not accompanied by splenic enlargement, the organ is usually nodular, and ascites is infrequent. *Amyloid disease* may be accompanied by splenic enlargement but not by jaundice. Enlargement of the liver may be associated with *chronic biliary obstruction*, but generally there is a history of recurrent attacks of colic and transient jaundice, and the spleen is not enlarged.

Prognosis.—As a rule the prognosis is unfavorable. The course of the disease extends over many years, but, unfortunately, ascites and the other symptoms due to portal obstruction, by which it is often first recognized, occur only as terminal events. Even at this period life may be much prolonged by judicious treatment. Should the disease be discerned earlier in its course, the outlook is more favorable. The hypertrophic stage is, therefore, sometimes amenable to treatment.

Treatment.—The cause, whether it be alcohol, or syphilis or some other infection, must if possible be discovered and removed. In order to reduce the activity of the liver, the patient should be placed on an exclusive milk diet. Only when the patient absolutely refuses to submit to this restriction is it advisable to permit other foods; and in such a case fats, sugars, and starches should be avoided and preference given to the proteid foods. The gastric catarrh should receive attention; in particular, the patient should sip a glassful of hot, preferably alkaline, water before each meal in order to free the stomach of mucus.

The early symptoms, which are principally gastro-intestinal, will afford indications for such drugs as *argentum nit.*, *arsenic*, *cinchona*, *iris*. *lycopodium*, *mercurius*, *nux vomica*, and *podophyllum*. Small doses of *mercurius* are recommended by physicians of all schools. Later, the appearance of ascites and the evidence of portal obstruction may lead to the selection of *aurum*, *iodine*, or *phosphorus*. *Potassium iodide* is

highly commended by many authorities, and is of undoubted value in cases of syphilitic origin.

As the disease progresses, purgatives and diuretics may be used to lessen the abdominal stasis. *Mercurius dulcis*, 1x, in grain doses every two or three hours, is often of great service at this stage. *Digitalis*, half a drachm or more of the insion every three hours, *caffein*, gr. iv, three times a day, *theobromine*, gr. v–x, t. i. d., may be used. Ultimately, however, paracentesis becomes necessary for the removal of the ascitic accumulation, and since the latter re-accumulates rapidly the operation may have to be repeated many times. After emptying the bladder with a catheter, introduce a trocar at a point in the median line midway between the umbilicus and the pubis. Withdraw the fluid slowly, and when the latter ceases to flow, remove the canula, cover the wound with adhesive plaster, and apply an abdominal binder. Under strict antisepsis the operation is free from danger.

Notable amelioration of the symptoms due to portal obstruction may be secured by surgical means. The parietal peritoneum and the omentum are scarified and then sutured together, and in this way anastomatic channels sufficient for collateral circulation are provided.

AMYLOID DEGENERATION OF THE LIVER.

(Lardaceous, albuminoid, or waxy liver.)

Etiology.—Amyloid degeneration of the liver, like amyloid disease of other organs, is usually the result of prolonged suppuration, notably that incident to tuberculosis, or of syphilis. Less often it is associated with cancerous and other cachexias.

Pathology.—The liver is enlarged, often enormously, but preserves its normal shape and smoothness. On section the tissue appears waxy and stains a mahogany brown with a watery solution of iodine. Similar changes are found in the other viscera, particularly the kidneys and the spleen.

Symptoms.—The degeneration occasions no characteristic symptoms, neither jaundice nor portal obstruction ensuing.

The patient complains of sensations of enlargement and weight in the hepatic region, and he becomes anemic, and finally dropsical. The associated amyloid change in the kidneys occasions the characteristic signs of that lesion.

Diagnosis.—The diagnosis rests upon the following data:

1. A history of the conditions favoring amyloid degeneration (prolonged suppuration, tuberculosis, syphilis, etc.).
2. An enlarged, firm, smooth liver, which is not tender.
3. An associated cachexia and often dropsy.
4. Evidences of amyloid degenerations in other organs.

Prognosis.—Should the causal condition be removable, the degenerative process in the liver may be arrested. In general, however, the amyloid change is progressive and terminates in the course of a few years in death.

Treatment.—Treatment must be directed to the primary disease. The suppurative lesion may be controlled by surgical means, the medicinal treatment being directed to the associated symptoms. Syphilitic cases should receive appropriate remedies, the more important of which are *aurum*, *mercury*, and *potassium iodide*.

ACUTE YELLOW ATROPY OF THE LIVER.

(Acute parenchmatous hepatitis; malignant jaundice; icterus gravis.)

Etiology.—The exciting cause of this rare and fatal disease is unknown, although it is probably a violent toxin, perhaps autogenetic or infective in its origin. The disease occurs more often in middle-aged women and rather frequently during pregnancy. Pre-existing infectious diseases and alcoholic and other excesses seem to predispose to it. A similar lesion may be produced by the toxic action of arsenic, antimony, or phosphorus.

Pathology.—The liver becomes small, its capsule is wrinkled, and microscopically the cells appear disintegrated into fat droplets and granules or crystals of bile pigment. The kid-

neys undergo parenchmatous inflammation, and the other viscera show degenerative changes.

Symptoms.—1. Prodomal stage. The early symptoms are those of gastro-intestinal irritation, including anorexia, nausea, vomiting, and prostration. Jaundice appears in a few days, beginning about the face and neck and extending over the whole body.

2. Stage of full development. In the course of a few days the prostration becomes more intense and the jaundice more marked, the skin even assuming a greenish hue. The temperature rises, and grave nervous symptoms—headache, delirium, convulsions, stupor, and coma—develop. Hemorrhages may arise from the skin or mucous membranes. At first the liver seems to be enlarged, but it rapidly diminishes in size and in a few days its dulness may be discoverable only in the axillary line. The urine is concentrated, deeply bile-stained, and slightly albuminous; the amount of urea is diminished, and casts, leucin, and tyrosin are present. The spleen is enlarged.

Diagnosis.—In the prodromal stage the disease cannot be distinguished from ordinary catarrhal jaundice, but the nervous symptoms, ecchymoses, hepatic atrophy, and urinary changes of the later stage render a diagnostic error improbable. *Phosphorus-poisoning* is accompanied by vomiting, violent gastralgia, and diarrhea, nervous symptoms are less pronounced, and leucin and tyrosin are not found in the urine. In phosphorus-poisoning a remission of three or four days follows the gastro-intestinal symptoms and precedes the appearance of jaundice. Phosphorus may be discovered in the stomach contents. *Yellow fever* may be distinguished by its geographical localization, and by its sudden invasion, which is accompanied by general pains, black vomit, and high fever.

Prognosis.—Death almost invariably occurs, generally within two weeks, as the result of cholemia or uremia.

Treatment.—A milk diet, cold water enemata, and the administration of *phosphorus* are advisable. Special symptoms may afford indications for other remedies.

FATTY LIVER.

Varieties.—An accumulation of fat in the cells of the liver may occur in two forms, viz.,

1. Infiltration of the cells with fat drops, which simply push the protoplasm to one side and thus by preventing its nutrition may ultimately cause its disappearance (*fatty infiltration*).

2. Metamorphosis of the cell protoplasm itself into fat, with consequent impairment of its function and ultimately complete cell disintegration (*fatty degeneration*).

Fatty infiltration of the liver may be due to :

(1) Over-ingestion of fats, which are stored in the liver; or

(2) Diminished oxidation, notably that attendant upon anemic and cachectic states, which permits the fat to accumulate unchanged in the liver. This is particularly common as a result of alcoholism and of pulmonary tuberculosis.

The fatty liver becomes greatly enlarged, smooth, and pale, and the microscope exhibits hepatic cells containing large drops of fat. Outside of the physical condition, which may be ascertained by palpation and percussion, the disease presents no characteristic symptoms. The liver can be completely restored to its normal state by control of the causal condition; otherwise, its unfavorable influence on digestion and general nutrition may hasten death from other causes.

Fatty degeneration of the liver is a grave disease, probably always toxic in origin, as a result of which the individual cells become degenerated and destroyed and the liver undergoes a reduction rather than an increase in size. The process, examples of which are seen in phosphorus-poisoning and acute yellow atrophy, is uncontrolled by treatment and invariably ends in death.

CANCER OF THE LIVER.

Etiology.—Carcinoma of the liver is usually secondary to cancerous growths of other organs, especially those related to the liver by the portal circulation. As a rule, though not

always, it attacks individuals who have reached middle or advanced life.

Pathology.—Cancer of the liver occurs chiefly in two forms viz.,

1. The nodular form. Multiple nodules, grayish white in color and varying in number from three or four to several hundred, are scattered through the substance of the organ, and may be felt upon its surface, even through the abdominal wall. Degeneration and absorption of the central cells of the nodule may be followed by contraction which leads to an umbilication of its centre.

2. The diffuse form. A general cancerous infiltration of the liver sometimes occurs and as a result the organ becomes enlarged in all its diameters.

Less frequently the cancer is radiating in form, and rarely it is colloid. In a few cases it is engrafted on a pre-existing cirrhosis and in consequence the liver becomes slightly if at all enlarged. Degenerative changes may lead to hemorrhages, and the extravasated blood may burst into the gall-bladder or peritoneal cavity. Occasionally suppuration occurs around a nodule.

Symptoms.—The patient shows signs of ill health; he complains of loss of strength and some *tenderness* or *pain* in the region of the liver, his appetite is poor and his digestion impaired, and he rapidly becomes emaciated and *cachectic*. *Jaundice* is present in nearly one-half of the cases, as the result of obstruction to the bile ducts. Ascites is infrequent, occurring only when the portal vein or its branches become obstructed by the growth. Physical examination reveals *enlargement* of the liver, which is often enormous; and frequently it is possible to palpate the superficial nodules and even their central umbilications.

Diagnosis.—In the presence of a recognized primary growth elsewhere the hepatic neoplasm may for a time escape observation, but ultimately attention will be directed to the liver and its enlargement noted. Advanced age, rapid emaciation,

hepatic tenderness, and the presence of either jaundice or ascites afford strong presumptive evidence of hepatic cancer. *Fatty infiltration* and *amyloid degeneration* are distingished by their history and by the absence of malignant symptoms and of jaundice. Multilocular *echinococcus cysts* are rare; they fluctuate, and are associated with splenic enlargement. *Tumors of adjacent organs* do not move with respiration. An *enlarged gall-bladder* is pear-shaped, and its situation is lower.

Neoplasms other than carcinoma, especially sarcomata, may involve the liver, but they correspond so exactly in both clinical course and prognosis that differentiation is unnecessary.

Prognosis.—The disease is invariably fatal, usually within a year.

Treatment.—Symptomatic and palliative.

JAUNDICE.

(Icterus.)

Etiology.—Staining of the tissues and fluids of the body with bile pigment is a symptom, not a disease. It is usually due to obstruction of the bile-ducts, the result of:

1. Catarrhal inflammation and swelling of the mucous membrane lining the duodenum or the ducts (catarrhal jaundice).
2. Foreign bodies (gall-stones, parasites) within the ducts.
3. Stricture or obliteration of the duct.
4. Tumors obstructing the duct from within.
5. Pressure on the duct from without (enlarged glands in the hilum of the liver, tumors of the adjacent viscera, displaced organs, fecal accumulations, cicatrices, gravid uterus, abdominal aneurism).
6. Lowered blood pressure in the hepatic circulation favoring resorption of the bile, as in the simple icterus of the newborn.

In addition to the above obstructive causes, the jaundice resulting from which has been termed *hepatogenous*, a second

variety of icterus has been ascribed to non-obstructive causes, such as widespread necrosis of the liver cells or increased destruction of the blood cells (yellow fever, phosphorus-poisoning, etc.), and termed *hematogenous*. The propriety of this distinction is questioned.

Symptoms.—1. *Cutaneous symptoms.* The *jaundice* appears first on the sclerotic or upon the mucosa of the mouth, especially when the latter is rendered temporarily anemic by pressure, and then upon the skin, first at its thinnest parts. The shade of yellow varies, and marked changes in its hue may be noticed from day to day. The patient is tormented with *itching* of the skin, which is apt to be worse at night. Erythematous or bullous eruptions, and if the disease be of long-standing, xanthelasma, may be associated; and in cases of grave icterus there may be ecchymoses.

2. *Intestinal symptoms.* Failure of the bile to reach the intestine (*acholia*) leads to *lessened digestive activity* and fat-absorption. The lack of bile wherewith to precipitate the pepsin from the chyme permits that substance to destroy the pancreatic ferment, and as a result the digestion of proteids and carbohydrates is also hindered, and general nutrition suffers accordingly. The patient is usually *constipated*, and putrefaction of the intestinal contents leads to gas-formation and consequent *meteorism*. The tongue is coated, there may be anorexia, nausea, and vomiting, and the feces are whitish and contain an excess of undigested fat. Less commonly an excess of bile pours into the intestine (*polycholia*) causing a dark coloration of the stools and perhaps a biliary diarrhea.

3. *Secretory symptoms.* Bile-pigments change the color of the urine to a yellow or brown which becomes greenish on exposure to the light. Albumin and casts may be present. The perspiration, the sputum, and even the breast milk may be more or less stained with bile. In any secretion its presence will be revealed by Gmellin's test. (See page 201.)

4. *Symptoms of auto-intoxication.* Various toxic phenomena may be observed. The *pulse is slowed* (40–50), the patient is

depressed in spirits and often irritable, progressive emaciation is apt to ensue, and such *grave nervous symptoms* as delirium, convulsions, tremors, and the typhoid state may appear. The temperature is lowered, and visual disturbances are not uncommon.

Diagnosis.—The sallow skin of general ill-health must not be mistaken for jaundice; it is usually an anemia and is not accompanied by straining of the conjunctiva and the secretions. *Addison's disease* presents a brownish discoloration which is more marked on exposed portions of the body and does not stain the sclerotic or the urine.

It is not enough, however, to recognize the existence of jaundice; diagnosis implies discovery of the cause and seat of the obstruction. Acute jaundice is usually due to catarrh of the common duct; if there is fever, the smaller ducts are involved. If the gall-bladder is distended and palpable, it suggests obstruction of the common duct, and if it is painful and tender, the obstruction is probably a gall-stone. The paler the feces, the more complete the obstruction and the more probable its seat in the common duct; for with obstruction of the cystic duct, bile still enters the intestine; and with obstruction of the hepatic duct, bile continues to flow until the gall-bladder is emptied and the jaundice is not associated with distention of the gall-bladder. Persistent jaundice indicates organic liver disease or permanent outside obstruction.

Prognosis.—This depends entirely upon the cause.

Treatment.—That of the causative condition.

ICTERUS NEONATORUM.

Diffuse jaundice is not unusual in new-born children, and two forms are recognized, the first of which is similar to ordinary obstructive jaundice and disappears in a week or two, while the second is usually fatal. This grave variety may be due to absence of the hepatic or common duct, to congenital syphilitic hepatitis, or to septic phlebitis of the umbilical vein. Treatment is unnecessary in the first variety and useless in the second.

SIMPLE CATARRHAL JAUNDICE.

Etiology.—Jaundice may be due to catarrhal inflammation of the common duct, not the result of an impacted gall-stone, and the local catarrhal condition may be caused by:

1. Local infection, the exciting cause of which is unknown. The disease may be epidemic.

2. Extension of an inflammatory process from the duodenum (gastro-intestinal disorders).

3. Infectious diseases elsewhere, especially pneumonia, typhoid fever, etc.

Pathology.—The mucous membrane of the duct becomes greatly swollen and its lumen may be occluded with mucus.

Symptoms.—1. *Jaundice*, with yellow skin, dark urine, and pale stools.

2. *Gastro-intestinal derangements*, with anorexia, nausea, vomiting, constipation, etc.

3. Slight *fever* may be present.

Diagnosis.—The appearance in a young adult of jaundice unaccompanied by pain or emaciation, especially if there is a history of pre-existing gastro-intestinal catarrh or other causative condition, usually justifies a diagnosis of catarrhal jaundice. Exceptionally it is necessary to exclude acute yellow atrophy, cancer, or cirrhosis.

Prognosis.—Favorable, recovery usually being completed within three weeks.

Treatment.—The patient should be placed at rest in bed and his diet limited to milk, beef broths, egg albumin, and an abundance of plain or saline water. Warm baths and weak solutions of carbolic acid may be used to relieve the itching of the skin.

The more important remedies, which are usually indicated by the gastro-intestinal symptoms, are *chelidonium*, *cinchona*, *colocynth*, *hydrastis*, *kali bichromicum*, *mercurius*, *nux vomica*, and *podophyllin*.

CHOLELITHIASIS.

(Gall-Stones; Biliary Lithiasis; Biliary Calculi.)

Etiology.—At least two-thirds of the cases of cholelithiasis occur in women, usually those past 40 years of age. Irregular meals, the excessive ingestion of carbohydrates and fats, tight-lacing, constipation, pregnancy, and the gouty diathesis have been named as predisposing causes. Most of these are conditions which interfere with the free flow of the bile, causing its retention in the gall-bladder or ducts. In addition, a catarrhal inflammation extending into the gall-ducts may partially occlude the passage with mucus and desquamated epithelium, thus slowing the current of bile. As a result, cholesterin, bile-pigments, and salts of lime and magnesia are precipitated about a nucleus composed of epithelium or mucus, and a gall-stone is formed. These calculi may vary in size from a grain of sand to a peach, and in number from a single one to several thousand; they range in color from yellow to brown or green, and are apt to be irregularly faceted.

Course.—1. The stone may remain latent in the gall-bladder or ducts.

2. The stone may pass out through the ducts; in that case its passage may or may not be accompanied by severe pain (*biliary colic*).

3. The stone may become impacted:

(*a*) In the cystic duct, causing distention of the gall-bladder with a thin mucoid fluid (*dropsy*, *hydrops vesicæ felleæ*) or with pus (*empyema*, *suppurative cholecystitis*). Ulceration and perforation may ensue.

(*b*) In the common duct, causing obstructive jaundice, biliary colic and sometimes intermittent fever.

4. The stone may ulcerate through into the peritoneal cavity, the adjacent viscera, or through the abdominal wall externally.

5. The stone may become impacted in the intestine, causing obstruction.

Symptoms.—1. *Calculi in the gall-bladder* may exist for an indefinite time without producing symptoms. In some cases the patient complains of occasional attacks of discomfort, tenderness, or pain in the hepatic region, with reflex digestive disturbances and sometimes slight fever.

2. *The passage of calculi through the ducts* may, if the stones are small, give rise to no symptoms; but the larger calculi occasion an attack of biliary colic severe in proportion to the size of the stone and the degree of obstruction which it causes. *Biliary colic* is characterized by a sudden attack of excruciating pain which centres in the right hypochondrium and is accompanied by nausea, vomiting, and often cold sweat and collapsic symptoms. If the stone is in the common duct the typical tenderness at the Mayo-Robson point, with pain radiating thence to the umbilicus, will be present. In half of the cases jaundice appears, usually within 24 hours. The duration of the attack varies from a few hours to a week or more. Symptoms of chronic obstruction may follow.

3. (*a*) *Obstruction of the cystic duct* may be caused by cicatrices or neoplasms as well as gall-stones. Physical examination may reveal some rigidity of the muscles in the right hypochondriac region, with sensitiveness or pain which is more marked when a deep breath is taken. Careful palpation usually discloses the smooth, oval, distended and fluctuating gall-bladder. Jaundice never results from obstruction of the cystic duct alone. Dropsy of the gall-bladder or an acute infectious inflammation of the gall-bladder (*acute cholecystitis*) may ensue. It is accompanied by paroxysmal pain in the right hypochondrium, nausea, vomiting, and more or less prostration; and suppuration (*suppurative cholecystitis*, *empyema of the gall-bladder*), gangrene, and perforation may result. Cholecystitis may also follow an acute infectious disease, notably pneumonia or typhoid fever. After an attack of the latter disease its specific organism is found quite constantly in the biliary passages.

(*b*) *Obstruction of the common duct* may be due to the presence of foreign bodies, cicatrices, or to compression from

without, as well as to gall-stones. The symptoms vary with the cause. When it is an impacted gall-stone, occlusion of the outlet leads to biliary colic. A stone in the common duct produces tenderness at a point two-thirds of the way from the tip of the ninth costal cartilage to the umbilicus (Mayo-Robson point), and the pain runs from the point toward the umbilicus. More or less jaundice is present. Should the impaction persist, a *catarrhal cholangitis* arises and is accompanied by fever of an intermittent type (*Charcot's*, or *hepatic*, *intermittent fever*) which strongly resembles malaria in its recurrent paroxysms of chill, fever, and sweat. In a few cases the cholangitis becomes *suppurative*, and as a result the fever is more septic in type and leads to death.

4. *Perforation of the gall-tracts* may be the result of infective gangrene or pressure-necrosis induced by the stone. Perforation into the peritoneal cavity will be followed immediately by symptoms of general septic peritonitis. The symptoms of penetration into the gastro-intestinal tract are very obscure and a diagnosis is rarely possible unless intestinal obstruction occurs. Occasionally free bile in the stools will suggest the possibility of such an occurrence. Perforation into the urinary tract may be indicated by the appearance of large quantities of bile, cholesterin, or hepatic calculi in the urine. The thoracic cavity may be invaded by a hepatic abscess which penetrates into the pleural cavity or lung, or the pus may burrow from the gall-bladder into the subphrenic space or mediastinum without involving the liver, ultimately rupturing into the pleura or lung; in the latter case the symptoms are those of septic pleurisy. A biliary fistula may penetrate the abdominal wall and discharge externally; usually it follows the course of the round ligament and appears at the umbilicus.

5. *Impaction of a gall-stone in the intestine* will give rise to symptoms of intestinal obstruction (*q. v.*). As a rule a stone large enough to become impacted in the bowel will first have ulcerated through from the gall-duct.

Diagnosis.—In the absence of distinctive symptoms the diagno-

sis of cholelithiasis may be difficult. Great significance should be attached to a history of attacks of colic, of recurrent jaundice, or of an intermittent fever, especially if typhoid, tuberculosis, and malaria can be excluded. Careful physical examination may detect a distended gall-bladder or other evidences of the lesion. Tenderness at the Mayo-Robson point accompanied by pain running from that point to the umbilicus is strong presumptive evidence of a stone impacted in the common duct. Obstructive jaundice arising in the common duct and accompanied by enlargement of the gall-bladder almost invariably means *cancer*. Obstructive jaundice thus arising without distention of the gall-bladder, almost always indicates a stone. (Van Lennep). Following a suspicious attack of colic, for several days the fecal discharges should be washed on a sieve in order to secure the positive evidence afforded by discovery of the stone.

Gastralgia usually occurs in neurotic individuals; the pain is referred to the stomach and back; it occurs when the stomach is empty, is relieved by eating, and fever and jaundice are absent.

Renal colic starts in the flank and the pain radiates down the ureter; the testicle of that side is painful and often retracted, and the urine may be scanty and blood-stained.

In *intestinal colic* the pain usually centres about the umbilicus and is relieved by firm pressure; often there is flatulence, and neither jaundice nor the stone can be discovered subsequently.

Prognosis.—Attacks of biliary colic usually terminate favorably; but the prognosis as to complete recovery must be guarded. If the inflammatory conditions are limited to the gall-bladder, if it is possible to remove all stones and to break up adhesions, perfect recovery may be assured; but generally this is possible only through surgical intervention.

Treatment.—During a paroxysm of biliary colic some relief may be obtained from the application of heat and the administration of symptomatically indicated remedies (*belladonna*, *calcarea*

carb., *nux vomica*); but since the cause of the pain is essentially mechanical, only an opiate can be expected to afford decided relief. *Morphine*, gr. ¼, with *atropine*, gr. $\frac{1}{120}$, may be given hypodermatically, and in aggravated cases inhalations of chloroform may be necessary. Evidences of persistent impaction, the development of dropsy or empyema of the gall-bladder, or suppurative symptoms of any kind justify surgical intervention. The surgical procedure in cases of cholelithiasis consists of removal of the calculi, followed by drainage or irrigation of the biliary passages to cure the catarrh.

When the attack has subsided, the patient should be placed on a diet free from alcohol, fat, sugar, and starch. He should live an active open-air life as far as possible; and if a prolonged visit to Saratoga or Bedford Springs is impossible, he should be ordered to drink one quart of water, containing from one to three drachms of Carlsbad Sprüdel salt, daily. Medicines should be selected in accordance with the indications present; the more important are *calcarea carb.*, *chelidonium*, *cinchona*, *lycopodium*, *nux vomica*, and *sulphur*.

Cancer, either primary or secondary to carcinoma of the liver or other adjacent viscera, may have its seat in the gall-bladder or gall-ducts. In addition to such general symptoms as emaciation and cachexia, evidences of biliary obstruction are usually present.

Stenosis of the common duct may be due to ulceration arising during the passage of a gall-stone, subsequent inflammatory adhesions completely occluding the duct. It may also be caused by external pressure, such as that of a tumor. Obstructive jaundice results.

Parasites (echinococcus, distoma hepaticum, etc.) may cause obstruction by their entrance into the larger biliary passages. The symptoms do not differ from those of hepatic obstruction due to other causes, except in cases where the presence of the echinococcus gives rise to a hydatid cyst.

DISEASES OF THE PORTAL VEIN.

Thrombosis of the portal vein (*adhesive pylephlebitis*), though rare, may result from traumatism, cirrhosis, cancer, external pressure, or retardation of the blood current; it is probable that a roughened venous wall is essential to the formation of the thrombus. It may occur in any portion of the portal tree, may be either partial or complete, and if organization of the clot ensues the vessel becomes transformed into a fibrous cord. Symptoms may be absent if the thrombosis is limited to portions of the hepatic vessels; if it is extensive, the sudden obstruction leads to the signs of venous stasis, *i. e.*, ascites, gastro-intestinal disorders, and hemorrhages. Jaundice is unusual. Hepatic cirrhosis can be excluded by a history of a sudden onset and the absence of atrophy of the liver. If the obstruction is limited to one of the smaller branches of the veins, an efficient collateral circulation may become established; but if the larger branches are occluded, the ultimate prognosis is highly unfavorable. The treatment should be that recommended for cirrhosis of the liver.

Septic thrombosis of the portal vein (*suppurative pylephlebitis*) is usually secondary to suppurative inflammations of the intestines (gastric ulcer, appendicitis, typhoid fever, dysentery) or to abdominal abscesses. Rarely it may be due to perforation of the portal vein by a foreign body (*e. g.*, a swallowed fish bone), and in new-born children it may follow septic infection of the umbilicus. Septic emboli are washed into the liver, where they lodge upon the walls of the larger vessels or within the minute hepatic branches and give rise to multiple abscesses. The symptoms are those of (1) abscess of the liver, together with (2) portal obstruction, and (3) general septicemia or pyemia. The condition is hopeless, and treatment can be only symptomatic or palliative.

DISEASES OF THE PANCREAS.

Diseases of the pancreas are by no means common, and even when they are present the accompanying symptoms are so obscure that their recognition is often extremely difficult. There may be discomfort or actual *pain* deep in the epigastrium, and should the solar plexus be implicated the pain may become severe and agonizing, radiating in every direction. Disturbance of the secreting function of the gland may lead to deficient pancreatic digestion, with *fatty stools* as a result. In such stools the fat appears as solid masses, white or yellow in color; sometimes it is liquid at the body temperature, and becomes solid on cooling after ejection. *Glycosuria* is frequenty the result of serious degenerative or destructive lesions of the pancreas.

The head of the pancreas is in close relation posteriorly to the common bile duct, the inferior vena cava, and the portal vein; and in consequence pancreatic tumors in that situation may press upon the bile duct, causing persistent *jaundice*, or by pressure upon the blood vessels it may bring about *edema* and *ascites*. Pyloric or duodenal obstruction may be induced in the same way.

Physical examination of the pancreas is very difficult, owing to the depth at which the organ lies in the epigastrium behind the stomach. Under normal circumstances it is completely out of reach, and only rarely do morbid changes render it more accessible. With the stomach and colon empty and the abdominal walls relaxed, deep palpation may sometimes enable the examiner to detect a tumor at a point about midway between the umbilicus and the ensiform appendix, which, in the presence of suspicious symptoms, may be attributed to the pancreas.

ACUTE PANCREATITIS.

Etiology.—The cause of this rare affection is practically unknown. It occurs oftenest in men over thirty, a few of whom

are alcoholic and many of whom have previously suffered with gastro-intestinal disorders. It may originate in a pancreatic hemorrhage induced by traumatism.

Pathology.—The pancreas becomes swollen and infiltrated with blood. White spots of fat necrosis may alternate with hemorrhagic foci, and the hemorrhagic effusion may involve the peri-pancreatic tissues and the mesentery (*hemorrhagic pancreatitis*).

Gangrene often follows these hemorrhagic extravasations, either the tip or the entire gland becoming softened and necrotic (*gangrenous pancreatitis*).

In some cases the lesion becomes purulent instead of gangrenous, and as a result the organ contains one or many small abscesses (*suppurative pancreatitis*). Localized peritonitis and thrombosis of the splenic and portal veins may ensue.

Symptoms.—The patient is suddenly attacked with severe and deep-seated pain in the epigastrium, and this is followed by vomiting, abdominal distention, and constipation. There is tenderness in the epigastric region, often a subnormal temperature, and usually the patient dies within three days. Should he survive longer, gangrenous changes may supervene, and are accompanied by chills, fever, sometimes jaundice, and finally collapse and death. Rarely life is prolonged over three or four weeks, and during that time irregular chills and a septic temperature may indicate that a suppurative process is in progress.

Diagnosis.—*Acute intestinal obstruction* generally occurs in the lower portion of the tract, is often accompanied by visible peristalsis of the intestinal coils and by fecal vomiting, often projectile in character; and it lacks the localized epigastric pain and tenderness of pancreatitis. *Irritant poisoning* may be excluded by the history, and by examination of the mouth and the vomitus. *Intestinal* or *gastric perforation* is almost always preceded by symptoms of ulceration. *Perforation of the gall-tracts* usually follows symptoms of cholelithiasis, the pain and tenderness are in the region of the gall-bladder, and jaundice is common.

Prognosis.—The disease is exceedingly fatal, death usually occurring within a week.

Treatment.—The treatment is largely palliative. Shock may be combated by the use of external heat, stimulants, and saline infusions. Symptoms may suggest the administration of *arsenic*, *belladonna*, *iris*, *mercurius*, or *phosphorus*. Surgical interference may be considered.

Chronic Pancreatitis, an intestinal fibrosis as the result of which the organ becomes hardened and the secreting structure atrophied and degenerated, is frequently found in connection with diabetes mellitus. In the absence of glycosuria it presents no characteristic symptoms. Associated digestive disturbances may afford indications for various remedies.

Pancreatic calculi may result from the precipitation of the inorganic constituents of the pancreatic juice. *Pancreatic colic* may accompany their passage through the duct. The symptoms of the latter resemble those of biliary colic, but the pain usually radiates to the left, and jaundice occurs only in those rare cases in which the calculus, becoming impacted near the orifice, presses upon the common bile duct. A positive diagnosis is, however, almost impossible, and the treatment must be symptomatic.

Pancreatic cysts arise from obstruction of the duct by calculi, cicatricial contraction, or pressure from without. The resulting retention-cysts may be single or multiple, and contain a viscid alkaline fluid. No symptoms present themselves until the objective evidences of an abdominal tumor appear. The latter is situated in the epigastrium or left hypochondrium, and is smooth, globular, and resistant. It may be difficult to distinguish it from other abdominal tumors. If aortic aneurism can be excluded, aspiration should be made. The fluid obtained from a pancreatic cyst is frequently capable of emulsifying fats and converting starch into sugar. The treatment is surgical.

Cancer of the Pancreas usually begins in the head of that organ, but its further course may involve the entire gland, converting

it into a symmetrical tumor; or it may be scattered through the organ in the form of cancerous nodules. The symptoms include digestive disorders, dull pain in the epigastrium, emaciation, and cachexia. The stools may be fatty, glycosuria may be observed, and obstruction to the common bile duct frequently causes jaundice. The disease usually proves fatal within a year. As a rule, the treatment is either symptomatic or merely palliative. Operation may be considered, however.

DISEASES OF THE SPLEEN.

Most of the morbid conditions of the spleen are associated with diseases of the blood, and its remaining lesions are, as a rule, secondary to disorders of other organs. Occasionally, however, attention is attracted to the latter by subjective or objective signs originating in the spleen. Enlargement of that organ may give rise to sensations of *dragging weight* in the left hypochondriac region (malaria, leucocythemia) or its rapid increase in size may exert traction on its own interstitial framework and thus cause *pain*. Pain may also result from inflammation of its peritoneal covering (*perisplenitis*).

Examination of the Spleen,—With the patient recumbent, lay the hand flat on the abdomen so that the finger tips, pushing up a fold of skin, lie close to and under the left costal margin at the 10th cartilage. If the spleen is greatly enlarged its edge can then be felt. If it is not, press forward with the other hand, placed posteriorly about the 10th rib, and let the patient take a deep breath; the sharp edge of the spleen, moving with respiration, can then be felt to ride over the fingers.

In order to percuss the spleen, have the patient carry his left arm up over his head. Then begin at the costal margin and percuss along the 10th rib until dulness is found; this represents the anterior border of the spleen, and normally it should be in the mid-axillary line. Percuss again from a point midway between the posterior axillary and scapular lines downward until resonance becomes impaired; this represents

the upper border of the spleen, normally about the 9th rib. Percuss along the 10th rib from the spinal column outwards; normally splenic dulness occurs about 1½ inches from the median line, but it is difficult to determine exactly. Percuss from below the border of the ribs in the posterior axillary line in order to outline the lower border of the spleen; normally it is just above the costal margin.

The area thus outlined is normally an oval a trifle more than 2 by 3 inches in size. Clinically, it is sufficient to determine the vertical diameter; if it exceeds 3½ inches, enlargement of the spleen may be at least suspected, but the diagnosis is not positive unless the lower edge is palpable.

The spleen becomes more or less acutely enlarged in connection with almost all of the acute infectious diseases. A chronic enlargement accompanies malaria, leucocythemia, splenic anemia, pernicious anemia, and amyloid disease; and it may result from portal congestion due to either cardiac or hepatic obstruction. The spleen may become displaced downward by a pleural effusion, emphysema, or a thoracic tumor. It may be pushed upward by tympanites or ascites. A floating spleen, recognizable by its shape, its notched edge, and its mobility, may be encountered in the abdominal cavity. (See *splanchnoptosis.*)

Splenitis may be the result of traumatism, or it may be due to extension of inflammation from an adjacent organ, such as the stomach, diaphragm, or lungs. It is accompanied by tenderness and enlargement of the organ.

Splenic Abscess occurs in connection with pyemic processes; it may rupture into the gastro-intestinal tract, the peritoneal cavity, or the lung.

Rupture of the Spleen may result from traumatism or from a sudden intense hyperemia of the organ, such as occurs in a pernicious malarial fever. It occasions sharp pain in the left hypochondrium, speedily followed by collapse and death.

Neoplasms of the Spleen are usually gummatous; occasionally carcinoma, sarcoma, or tuberculosis involves the organ; and a hydatid cyst is possible.

Amyloid Spleen is indicated by the discovery of a hard, smooth enlargement of the organ, associated with amyloid degeneration of other organs and a history of its causative conditions.

Floating Spleen may be discovered as a movable hard tumor of characteristic shape in the abdominal cavity. It should be restored to its place and retained there, if possible, by external support (see *splanchnoptosis*).

DISEASES OF THE BLOOD.

Attention may be directed to the condition of the blood by *pallor* of the skin and mucous membranes, associated with more or less *physical weakness*. The blood changes of which these symptoms are indicative may be *primary* (*essential anemia*), but in a great majority of cases they are *secondary*, *i. e.*, *symptomatic* of one of those visceral diseases which are accompanied by wasting (*e. g.*, tuberculosis, carcinoma, etc.). Only when the latter can be excluded is the diagnosis of a specific blood disease made possible by examination of that fluid, but since certain general diseases also lead to more or less characteristic hemic changes, a knowledge of the latter is frequently essential for purposes of differential diagnosis.

CLINICAL EXAMINATION OF THE BLOOD.

By examination of the blood we may determine:

(1) The existence or non-existence of a condition of anemia, and if the latter be present, its type and severity.

(2) The existence or non-existence of leucocytosis, and if that condition exists, its type and extent, including the recognition of leucocythemia.

(3) The presence or absence of blood parasites, including those pathognomonic of malaria, filariasis, and relapsing fever.

(4) The existence of definite serum reactions. such as the Widal test for typhoid fever. (See page 30.)

(5) The presence of bacterial organisms. For the identification of the latter, culture methods are necessary.

The practical examination of the blood includes investigation of each of the following details:

1. The macroscopic appearance of the blood.

2. The microscopic appearance of the blood, including—

(*a*) The appearance of the fresh blood.

(*b*) The appearance of the stained blood.

(*c*) The enumeration of the corpuscles.

(3) The estimation of the amount of hemoglobin.

To secure a specimen of blood, cleanse and dry the finger tip or the lobe of the ear, and prick it with a sharp needle or guarded lancet-point deep enough for the blood to exude of its own accord—pressure must not be used. Wipe away the first drop. The second and succeeding drops may be secured, as will be directed, for the various steps of the examination.

1. **Macroscopic appearance of the blood.**

Freshly shed blood appears to the naked eye as a homogeneous red fluid, which after a varying period of time separates into serum and clot. In certain pathological states (*e. g.*, hemophilia) it may be important to ascertain the time required for this coagulation to take place. For a rough clinical test, allow a few isolated drops of blood to fall on a glass slide. Draw a fine clean needle through one drop after another at short but regular intervals, and note the time which elapses before a filament of fibrin, drawn out from the drop by the needle, indicates beginning coagulation. More accurate results may be obtained by the use of Wright's coagulation tubes.

2. **Microscopic appearance of the blood.**

(*a*) *Examination of the fresh blood under the microscope.*

Touch the exuding drop with the cover-glass and immediately place the latter face down upon the slide, without exerting undue pressure. The specimen may be ringed with vaseline to prevent rapid drying out. Observe:

(1) *The shape of the red corpuscles* (*erythrocytes*).

Normally, under the microscope they appear disc-shaped with a central depression. Their contour is unaltered in chlorosis and other mild anemias, but in severe anemias they may appear to be distorted (*poikilocytes*), abnormally small (*microcytes*), or abnormally large (*macrocytes*).

(2) *The tendency to rouleaux formation.*

This disappears when the number of red corpuscles is excessively diminished, as in some anemias.

(3) *The number of red corpuscles.*

This can be determined accurately only by use of the counting apparatus, but the practiced eye will quickly recognize any great diminutition in their number.

(4) *The color of the red corpuscles*, normally yellow, may become notably pale, especially in cases of chlorosis.

(5) *The number of white corpuscles, especially in proportion to the red.* Normally there is one white to about five hundred red. This proportion becomes greatly increased (leucocytosis) in many diseases, and in some cases, especially in those of leucocythemia, this change may be discovered at a glance. As a rule, however, the counting apparatus is necessary.

(*b*) *The examination of the stained blood under the microscope* requires the preparation of films. For this purpose, allow a drop of blood to fall upon a clean, dry cover-glass and cover it with another glass, and then separate them by gently sliding one over the other. Or, allow the drop of blood to touch the surface of a perfectly clean slide and use the edge of another slide, held at an angle of about 45°, to spread the drop evenly along the slide. The films must be made quickly, before the corpuscles can become altered, and must be as uniform and as thin as possible. After allowing them to dry in the air, place the films for five minutes or more in a mixture of equal parts of absolute alcohol and ether, and then stain them from two to eight minutes in Ehrlich's triple stain; or, stain them first in an alcoholic solution of eosin for about half a minute, wash, and counter-stain with Delafield's hematoxylin solution (freshly filtered) for fifteen seconds or more. Wash in clean water, clear with xylol, and if a permanent preparation is desired, mount in Canada balsam. Then place one under the microscope, and search for—

(1) *Nucleated red corpuscles* ("*blasts*"). These are present in the blood during intra-uterine life, but appear after birth

only in connection with grave forms of anemia, and in leucocythemia. As regards size, they may be normal (*normoblasts*), abnormally small (*microblasts*) or abnormally large (*megaloblasts*). These normoblasts and megaloblasts may be distorted in shape (*poikiloblasts*).

(2) *The different forms of white corpuscles.* These are distinguished according to the character of their nuclei, as

a. Small mononuclear leucocytes, somewhat less in diameter than a red corpuscle, with a single large nucleus which occupies nearly the whole of the cell, and stains deeply with hematoxylin. Normally they constitute about 25% of the total number of leucocytes.

b. Large mononuclear leucocytes, often two or three times the size of a red corpuscle, with a large oval nucleus which stains but faintly with hematoxylin. They constitute about 6% of the leucocytes. A form in which the nucleus becomes indented, or horse-shoe-shaped, has been termed *transitional.*

c. Polynuclear leucocytes (*neutrophiles*), larger than the red cells, are characterized by several nuclei connected by threads so as to resemble one irregular nucleus. They constitute 65 to 70% of the leucocytes. This is the variety chiefly present in pus, and they are supposed by their action as phagocytes to protect the organism against bacterial invasion.

d. Eosinophile cells are large, round, and nucleated, and are characterized by glistening granules which become deeply stained by eosin. They constitute not over 2–4% of the leucocytes in normal blood.

e. Myelocytes (*Markzellen*).—Unlike the other leucocytes, these forms are never found in normal blood. They are large bodies, from three to seven times the diameter of a red corpuscle, and have an extremely pale nucleus lying in the centre or to one side of the cell. They differ from the large mononuclear or transitional forms in their much greater size and in the pallor of the nucleus.

The leucocytes may be also differentiated, on the basis of their staining reaction with a triple stain, into:

a. Basophiles, cells taking a blue stain in their protoplasm.

b. Neutrophiles, cells taking a lilac stain in their protoplasm.

c. Oxyphiles, cells taking a golden-red stain in their protoplasm.

Of the leucocytes, 20–25% are basophiles, 60–75% are neutrophiles, and ½–5% are oxyphiles.

A **differennial count** of the leucocytes is made by counting, say, five hundred or one thousand and classifying them according to the variety of their nuclei. This serves to indicate whether each is present in its normal proportion.

(*c*) *The enumeration of the corpuscles.*

(1) *Counting the red corpuscles.* For this purpose the Thoma-Zeiss apparatus is used. Place the point of the small

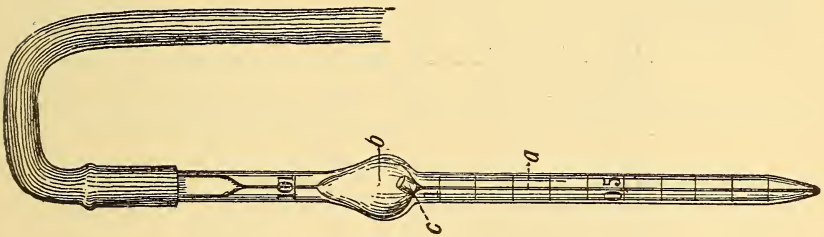

FIG. 23.—Thoma-Zeiss pipette..

pipette in a drop of fresh blood as it exudes from the puncture, and suck gently on the rubber tube until the blood exactly reaches the point marked 1; then quickly wipe the outside of the pipette with a clean cloth, to remove any adherent blood, and immerse the tip in the diluting fluid (Gower's solution, consisting of sodium sulphate, grs. 104; acetic acid, ʒj; and distilled water, ʒiv.) and suck until the latter reaches the mark 101. While doing this roll or shake the pipette, in order to thoroughly mix the fluids. Be careful lest the blood coagulate before the diluting fluid is drawn up, or an air bubble be drawn in through allowing the point of the pipette to slip out of the blood drops, and also lest by pressing the point of the pipette through the drop against the skin the column be prevented from rising.

The dilution of the blood is now 1 to 100. The counting chamber, a disc of glass on which parallel lines are engraved to form 400 squares each $\frac{1}{20}$ millimetre in length, is divided by double lines into groups of 16 squares. The depth when a cover glass is applied is $\frac{1}{10}$ of a millimetre.

Blow out three or four drops from the pipette, and then deposit a single small drop in the centre of the ruled disc and

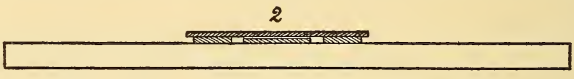

FIG. 24.—Blood-Counting Slide (Elevation).

apply the cover glass accurately, so that no air bubbles are enclosed, and so that Newton's rings can be seen.

Place the slide on the microscope in a horizontal position, and, using about a ¼ inch objective, count the number of red corpuscles found in several of the groups of 16 squares each.

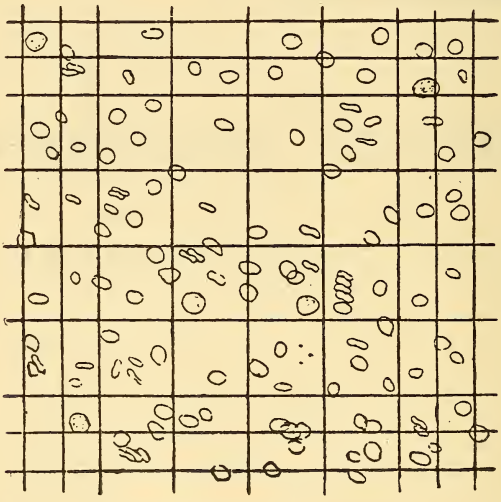

FIG. 25.—The appearance of the blood under the microscope, showing sixteen small squares enclosed by extra vertical and horizontal lines.

In counting, include those corpuscles seen on the lower and left lines, but omit those seen on the upper and right lines. To

calculate, remember that each square has a cubic capacity of

$$\frac{1}{20} \times \frac{1}{20} \times \frac{1}{10} = \frac{1}{4000} \text{ c.m.m.}$$

and therefore the formula for calculation must be

$$\frac{\text{No. of corpuscles counted} \times \text{dilution (100)} \times 4000}{\text{number of squares counted.}} = \text{number of corpuscles per c.m.m. of blood.}$$

In healthy men the red corpuscles are present to the number of about 5,000,000 per cubic millimetre, and in women about 4,500,000 per c.m.m. In chlorosis the number is little changed, but in the other anemias it is often much diminished.

(2) *Counting the white corpuscles.* This may be done with the special pipette, the diluting solution being a ⅓% solution of glacial acetic acid in water (to destroy the red corpuscles) with a small quantity of gentian violet added (to color the leucocytes). In this case the dilution is but 1 to 10; with this exception, the calculation is made from the count exactly as in the enumeration of the red blood corpuscles, viz.:

$$\frac{\text{No of corpuscles} \times \text{dilution (10)} \times 4000}{\text{Number of squares counted}} = \text{number of leucocytes per c.m.m. of blood.}$$

This method requires, on account of its larger pipette, such a quantity of blood that it is sometimes objectionable; and in consequence the ordinary pipette is often used for both counts.

In health the number of white corpuscles varies from 5,000 to 10,000 in every c.m.m. of blood. An increase of this number (*leucocytosis*, *hyperleucocytosis*) occurs physiologically during the digestion of proteids, and pathologically in many infectious and other diseases. A decrease (*hypoleucocytosis*) occurs in certain other diseases, notably typhoid, tuberculosis, and malaria.

Leucocytosis (*hyperleucocytosis*).—The causes of leucocytosis are many. It occurs physiologically in the new-born, in pregnant women, and after the ingestion of food. It occurs pathologically in all the infectious diseases except tuberculosis, typhoid, malaria, influenza, and leprosy. Streptococcic and

staphylococcic infections occasion a moderate increase of the number of leucocytes (15,000–40,000). Malignant tumors, especially sarcomata, the other cachectic conditions, and loss of blood by hemorrhage may give rise to a leucocytosis, and it may also be induced by various poisons (pilocarpine, camphor), certain bacterial toxins (tuberculin), etc. Leucocytosis is a symptom, not a disease, and of itself occasions no symptoms. When it exceeds 50,000 the existence of leucocythemia may be suspected.

Hypoleucocytosis (*leucopenia*). — Decrease of the number of leucocytes (below 6,000) occurs notably in tuberculosis when

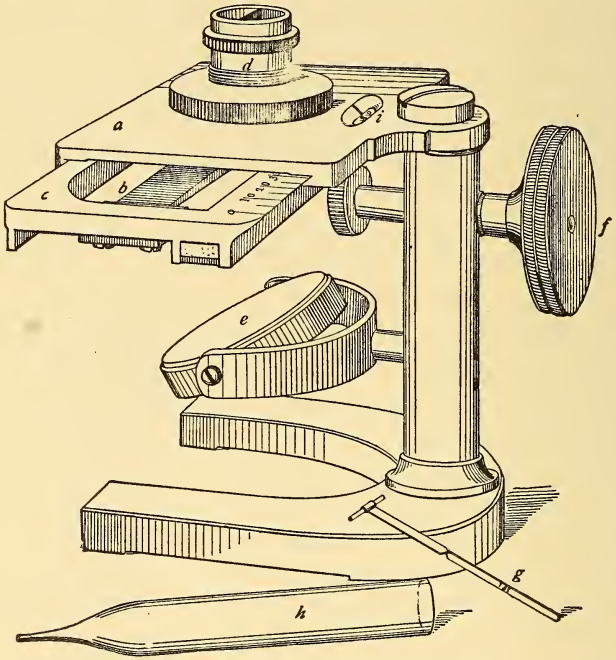

FIG. 26.—Von Fleischl's Hemometer.

no secondary infection has occurred and in typhoid fever; in cases where these diseases are suspected its discovery may have a decided diagnostic importance. In pneumonia an increase of leucocytes is the rule, but in some cases there is a

decrease, and the latter constitutes an unfavorable prognostic sign. Hypoleucocytosis is also found in the chronic symptomatic anemias, in cases of malnutrition, etc., but is without direct clinical significance.

3. **The Estimation of the Amount of Hemoglobin** is usually made by Von Fleischl's method, using the latter's hemometer (Fig. 26). The small capillary tube which accompanies the apparatus, held by the stem (*g*) in which it is fixed, should be brought in contact with an exuding drop of blood, when it immediately fills by capillarity. Quickly clear the outside of any adherent blood and place it in the water of one compartment of the cylinder (*d*), moving it about to dissolve out the coloring matter. Then, holding it vertically, allow drops of water from the pipette (*h*) to wash it clean, mix the blood and water thoroughly with the handle of the tube (*g*), and finally add water until the compartment is quite full. The other side, filled with water only, is placed over the wedge of colored glass (*b*). Artificial light, and as little of that as possible, is reflected upward through the cylinder, and the colored glass is moved backwards and forwards by jerks, until the tints of the two compartments appear to be about the same; then the percentage is read on the scale (*i*).

Tallqvist's Method.—This simple method for estimating the amount of hemoglobin requires only a Tallqvist hemoglobin book, which contains specially prepared porous paper to soak up the blood, and a color table. The latter consists of ten color plates, the upper one representing the color obtained by soaking the porous paper in blood containing but 10% of hemoglobin, the next plate a facsimile of the color obtained by the use of blood containing 20%, and each succeeding plate representing the color effect of a blood 10% richer, up to the normal 100%. To perform the test, secure a drop or two of blood on the porous paper, allow it to dry until its gloss disappears, and then compare it with the color table. That plate to which the shade of color corresponds indicates the percentage of hemoglobin.

In chlorosis the amount of hemoglobin is greatly diminished,

without notable change in the number of red corpuscles. In the other anemias the amount of hemoglobin is decreased in proportion to the diminution in number of the red cells.

SYMPTOMATIC ANEMIA.

Etiology.—Anemia may be secondary to:

1. *Hemorrhage.*—Loss of blood, either from traumatism or in the course of a disease, produces an anemia which is severe in proportion to the amount and rapidity of the blood loss.

2. *Environment, Food, etc.*—Insanitary surroundings and an unwholesome diet, by depressing the general health, have a marked effect upon the nutrition of the blood.

3. *Parasites.*—The malarial parasite, certain gastro-intestinal parasites, and the filaria sanguinis hominis are capable of inducing anemia by their interference with nutrition.

4. *Infectious Diseases.*—Anemia may be symptomatic of either an acute or a chronic infective process.

5. *Intoxications.*—Toxins produced by gastro-intestinal disorders, or poisons from without (lead, mercury, etc.) may induce blood destruction.

6. *Organic Diseases and Neoplasms.*—Diseases of the stomach, kidneys, lungs, and heart, and malignant tumors, may occasion anemia.

Pathology.—In such a case examination of the blood reveals:

1. Reduction of the amount of hemoglobin (from 75 to as low as 15%).

2. Red corpuscles of irregular form, sometimes microcytes and megalocytes, and if the anemia is severe, nucleated red corpuscles.

3. The proportion of leucocytes is often unchanged; sometimes they are increased in numbers, and rarely they are decreased.

4. Chemical changes. The proportion of albumen is decreased and that of water is increased.

Symptoms.—Among the symptoms suggestive of secondary anemia are:

1. *Pallor* of the skin and mucous membranes. This may be slight, or it may amount to a deathly whiteness; in some cases the skin is yellow or gray in color.

2. *Emaciation.* This is usually moderate in degree, and depends largely on the character of underlying conditions.

3. The *skin* is frequently dry and harsh.

4. *Gastro-intestinal disorders.* Poor appetite, digestive disturbances, constipation, etc., usually accompany the anemia.

5. *Circulatory disorders.* A rapid, weak heart, anemic murmurs, palpitation, dropsy, and serous effusions are common in anemic states.

6. *Physical weakness* and languor.

7. *Neuralgias.* These may, however, be due to the underlying cause.

8. *Dyspnea.* Occasionally this is accompanied by dry cough.

9. *Menstrual disorders.* Dysmenorrhea, amenorrhea, and rarely menorrhagia may occur.

Diagnosis.—It is necessary to distinguish a symptomatic anemia from *chlorotic anemia*, in which there is a lack of the causal conditions enumerated. It occurs particularly in young girls, and is accompanied by a loss in hemoglobin entirely out of proportion to the decrease in number of the red corpuscles.

Pernicious anemia is characterized by the presence of certain varieties of corpuscles (microcytes, macrocytes, poikilocytes, nucleated erythrocytes).

Prognosis.—This varies according to the underlying cause and the possibility of its removal.

Treatment.—1. Discover and treat the underlying affection.

2. Secure proper surroundings, sufficient hours of rest, and appetizing food.

3. Administer such remedies as *arsenicum*, *cinchona*, *iron*, etc.

4. A severe post-hemorrhagic anemia may sometimes require the use of saline infusions.

CHLOROSIS.

Etiology.—This is a primary anemia, occurring in young girls at puberty, to which heredity and improper surroundings seem to predispose. Older women and males are rarely affected. The exact cause is uncertain; it has been attributed to:

1. Undeveloped heart and blood vessels, in some cases (Virchow).

2. Infantile uterus and ovaries.

Pathology.—1. The blood is pale and abnormally fluid.

2. The amount of hemoglobin is markedly reduced (60–20%).

3. The red corpuscles are usually diminished but in lesser degree (4,000,000–1,500,000).

Symptoms.—The patient, usually a young girl, presents:

1. *Pallor* of the mucous membranes and skin, the latter often becoming greenish or yellowish in color.

2. *Digestive disorders* (poor appetite, defective digestion, constipation, etc.).

3. *Circulatory symptoms* (palpitation of the heart, sometimes edema, and dyspnea, due to the deficiency of oxyhemoglobin).

4. *Menstrual disorders*, especially amenorrhea.

Physical Examination.—1. *Blood changes.* There is no especial diminution in the number of erythrocytes, but a marked deficiency in the amount of hemoglobin.

2. *Anemic murmurs* may be heard over the heart, and possibly dilatation of the latter may be detected. These murmurs are usually systolic in time, and are heard most frequently over the pulmonary area; but they may also present themselves at the apex, over the tricuspid area, and at the aortic

orifice. A venous hum (*bruit de diable*) is heard over the root of the jugular vein.

Prognosis.—The disease tends to spontaneous recovery in a few weeks, and prompt improvement under treatment is the rule. There is danger of relapse, however, and also of such serious complications as gastric ulcer and pulmonary tuberculosis. Various cardiac, digestive, and nervous disorders may supervene.

Diagnosis.—The age and sex of the patient, together with the marked disproportion in the decrease of hemoglobin as compared with that in the number of corpuscles, usually renders diagnosis easy.

Treatment.—Personal hygiene. Rest, fresh air, and wholesome food are necessary. *Avoid constipation.*

2. Medicines. *Iron* is the principal remedy; less often *arsenic*, *manganese*, *sulphur*, and other medicines are required.

PERNICIOUS ANEMIA.

Etiology.—This is a primary anemia of obscure origin. Gastric atrophy, intestinal parasites, and infective diseases have been credited with its production; and it may be secondary to oral sepsis, *e. g.*, prolonged suppuration about a diseased tooth.

Pathology.—Blood destruction is progressive, with a diminishing number of erythrocytes (even down to 150,000) and some reduction of the hemoglobin, but not proportionately. The number of leucocytes remains approximately normal. The liver becomes enlarged, fatty, and pigmented with iron; the bone marrow undergoes cellular hyperplasia, and degenerative and hemorrhagic changes occur throughout the viscera.

Symptoms.—Pernicious anemia is characterized by :

1. *Pallor* of the skin, which assumes a lemon-yellow tint. There is no emaciation.

2. *General anemic symptoms.* Weakness, digestive disturbances, dyspnea, palpitation, syncope, edema, and hemic murmurs may be present.

3. The *urine* is of low specific gravity, and dark with pathologic urobilin.

4. *Febrile* attacks develop occasionally during the course of the disease.

5. *Blood changes.* The blood appears thin, pale, and watery, like "meat washings." On microscopical examination there is found to be:

(*a*) A reduction in the number of red corpuscles to 2,000,000 or below.

(*b*) Megalocytes, microcytes, poikilocytes, and nucleated red corpuscles are present.

(*c*) The amount of hemoglobin is not proportionately decreased.

(*d*) The leucocytes are approximately normal in number.

Prognosis.—The outlook is generally unfavorable; an apparent recovery is often but temporary.

Treatment.—Should any focus of suppuration be found about the teeth, secure oral antisepsis by the use of carbolic acid (1 in 40) or, better, let a dental surgeon remove all diseased tissue. Administer easily assimilable food (beef juice, peptonized milk) freely, and prescribe *arsenic* or such other remedies as may be symptomatically indicated. Febrile paroxysms may suggest the advisability of using *antistreptococcic serum.*

LEUCOCYTHEMIA.

(Leukemia.)

Etiology.—This is probably a disease of the blood-forming organs. Its exact cause, however, is unknown. It may be an infection.

Pathology.—1. The spleen becomes enlarged and adherent to adjacent structures.

2. Lymphatic enlargements occur; the mesenteric, cervical, inguinal, axillary and other glands may become increased in size.

3. The bone-marrow becomes "pus-like," owing to the increased number of leucocytes.

4. The liver, kidneys, and other organs undergo degenerative changes, and whitish nodules (consisting of proliferated leucocytes) may be found in them.

Symptoms.—The patient presents—

1. *General anemic symptoms* (pallor, gastro-intestinal disturbances, dyspnea, etc.).

2. Subjective *discomfort* due to the tumor (enlarged spleen) in left side of abdomen.

3. *Hemorrhages* from the mucous membranes (nose, intestines, lungs, kidneys), or retina.

4. *Fever* of irregular type with long intermissions.

5. *Lymphatic enlargements*, the glands being soft and movable and producing no symptoms except by pressure upon adjacent organs.

6. *Blood changes.* The blood may be simply pale, or it may even appear like pus, owing to the large number of leucocytes present. Microscopically it shows:

(*a*) An enormous increase in the number of leucocytes (up to 600,000) so that in proportion to the red cells they are as 1 to 50, 1 to 25, or even 1 to 2 or 3. The increase may be in the number of lymphocytes (*lymphatic leukemia* or *lymphemia*), but more commonly it is the myelocytes and eosinophiles (*spleno-medullary leukemia*) which are increased.

(*b*) The red corpuscles are somewhat diminished in number.

(*c*) The proportion of hemoglobin is reduced.

(*d*) Charcot's octahedral crystals appear in the blood if it is allowed to stand for a time.

Diagnosis.—This is rendered possible by the blood examination alone. An excessive *leucocytosis* is marked by an increase of the polynuclear neutrophiles only.

Course and Prognosis.—The lymphatic form may be acute, proving fatal within two months. Other cases, while less rapid, usually terminate in death within two years.

Treatment.—Attend to the patient's hygiene and nutrition, and administer such remedies as *arsenic*, *phosphorus*, *picric acid*, and *thuja*. Inhalations of oxygen have been recommended, and extracts of bone marrow may be tried. Various symptoms, such as those due to digestive disturbances, constipation, and diarrhea, will present indications for special remedies.

SPLENIC ANEMIA.

Etiology.—The cause is unknown.

Pathology.—Splenic anemia is distinguished by:

1. Great enlargement of the spleen.
2. Absence of enlargement of lymphatic glands.
3. Absence of leucocytosis.

Symptoms.—The course of splenic anemia is attended by:

1. *Enlargement of the spleen* and attacks of severe pain in the splenic region.
2. *Fever* of hectic type, reaching 102° or more in the evening.
3. *General anemic symptoms* (weakness, digestive disturbances, palpitation, dyspnea, etc.).
4. *Hemorrhages* from the mucous membranes.
5. *Blood changes* of chlorotic type, *i. e.*, the red corpuscles are reduced in number, with an even more marked diminution in the amount of hemoglobin.

Diagnosis.—*Leucocythemia* is distinguished by a remarkable increase in the number of leucocytes. *Pernicious anemia* presents characteristic blood changes without splenic enlargement. In *Hodgkin's disease* the anemia is not so profound, while the lymphatic glands are involved. *Malignant disease* of the spleen is marked by progressive emaciation and secondary growths elsewhere, without pyrexia. *Syphilitic disease* of the spleen may be distinguished by its history, associations, and by the therapeutic test.

Prognosis.—Hopeless; death from asthenia occurs within two years.

Treatment.—This must of necessity be symptomatic.

HODGKIN'S DISEASE.

(Pseudo-Leukemia; Lymphatic Anemia; Lympho-Sarcoma.)

Etiology.—The exciting cause is unknown.

Pathology.—Pseudo-leukemia presents the following lesions:

1. Hyperplasia of the lymph glands: The cervical lymphatics are the first to become enlarged, and the axillary and inguinal glands are next affected. Suppuration in these enlarged glands is a rare occurrence.

2. Hypertrophy of the spleen occurs in 80 per cent. of the cases.

3. New formations of lymphatic tissue may appear in any tissue of the body, especially in the liver, kidneys, lungs, and heart.

Symptoms.—Hodgkin's disease is characterized by:

1. *General anemic symptoms.* (Languor, dyspnea, palpitation, digestive weakness, etc.).

2. *Enlargement of the lymphatic glands*, usually detected first in the neck. Later, when they have increased in size, they project as large bunches. Subsequently similar enlargements are found in the axilla and groin.

3. *Enlargement of the spleen.* This gives rise to a sensation of heaviness or dragging, or even actual pain. The enlarged organ may be grasped through the abdominal walls.

4. *Febrile paroxysms* of irregular type. These often coincide with the involvement of new groups of glands.

5. *Blood changes.* At first the blood is practically normal, but later there is an anemia of symptomatic type.

Diagnosis.—The diagnosis rests upon the discovery of the lymphatic enlargements, unassociated with any characteristic blood changes. *Tuberculous glands* are found in younger

persons, are generally unilateral, and tend to suppurate, while the spleen and liver are not so markedly enlarged. *Syphilitic enlargement* of the glands may be distinguished by the history, the associated symptoms, and the therapeutic test. *Leucocythemia* presents a striking increase in the number of leucocytes.

Prognosis.—This is very unfavorable; death from exhaustion usually occurs within three years.

Treatment.—The patient's surroundings should be made as hygienic as possible, and his food must be nutritious and easily digestible. Surgical removal of the glands should be considered early in the disease, in the hope of thus arresting its progress. The principal remedies are *arsenic*, *arsenic iodide*, *iodine*, *iron*, *mercurius biniod.*, and *phosphorus*.

ANEMIA INFANTUM PSEUDO-LEUKEMICA.

Etiology.—This rare affection occurs in chilren under the age of four, and especially between the seventh and twelfth months of life. Rickets, hereditary syphilis, and gastro-intestinal disturbances seem to predispose to it.

Pathology.—The spleen becomes increased in size, the liver and lymphatic glands are often enlarged, and the bone marrow may be reddened and soft.

Symptoms.—1. The *spleen is enlarged* and may be distinctly felt projecting from beneath the ribs into the abdomen. It is extremely hard.

2. *Pallor*, weakness, emaciation, and other evidences of anemia are present.

3. *Blood changes*. (*a*) The red corpuscles are reduced in number below 3,000,000. Poikilocytes and erythrocytes are common.

(*b*) Marked leucocytosis is present, and the polynuclear leucocytes may be increased to 100,000 or more.

Diagnosis.—*Leucocythemia* may be excluded by discovery of the fact that the increase is in the number of polynuclear

leucocytes. The lack of hepatic enlargement and of glandular involvement, the co-existing evidences of rickets or hereditary syphilis, and the comparatively benign course also serve to distinguish the disease.

Prognosis.—The anemia is progressive, but under proper treatment it tends to recovery.

Treatment.—Pay close attention to the general hygiene of the patient, see that the child has a suitable nutritious diet, and prescribe such remedies as *arsenic*, *iodide of arsenic*, *iodide of iron*, and *phosphorus*.

HEMOPHILIA.

(Hemorrhagic diathesis; Bleeder's disease.)

Etiology.—Heredity appears to be the only important predisposing cause of hemophilia.

Pathology.—It is undecided whether the bleeding is due to chemical changes in the blood or to changes in the vessel walls.

Symptoms.—In a "bleeder," profuse and persistent hemorrhage follows trivial injury or even occurs spontaneously. It usually is limited to a capillary oozing, which may occur from or beneath the skin or mucous membranes or into the joints, and is often uncontrollable.

Diagnosis.—The tendency to uncontrollable hemorrhages usually appears in childhood, and inquiry often reveals the family predisposition.

Prognosis.—In many cases hemorrhage proves fatal in early life. If the child survives this danger, the tendency may be outgrown.

Treatment.—The treatment should be largely prophylactic; the child ought to be guarded against the slightest traumatism. He should be carefully nourished, and at the same time hardened by a judicious out of door life. Attacks of hemorrhage must be met by surgical measures, the subsequent treatment being that of secondary anemia.

PURPURA.

Purpura is a generic term used to designate a number of conditions characterized by hemorrhages into the skin.

Varieties.—1. PRIMARY PURPURA includes:

(*a*) *Simple purpura;* this is a form characterized by petechial or ecchymotic extravasations into the skin, the cause of which is unknown.

(*b*) *Arthritic purpura* (*Purpura rheumatica, Peliosis rheumatica, Schönlein's disease*) is a form characterized by swollen and painful joints, a purpuric eruption, and edema of the feet and ankles. It is accompanied by mild fever (101° to 103°). Severe gastro-intestinal disturbance may be associated (*Henoch's purpura*). The prognosis is favorable provided no complications (endocarditis, intestinal hemorrhage, hemorrhagic nephritis) arise.

(*c*) *Hemorrhagic purpura* (*Morbus Weilhofii*) is a form characterized by extensive subcutaneous ecchymoses and hemorrhages from the mucous membranes of the respiratory and gastro-intestinal tracts, or into the brain, kidneys, or serous cavities. It is accompanied by fever and prostration, and is often fatal.

2. SECONDARY PURPURA may be symptomatic of:

(*a*) *Infectious diseases*, including cerebro-spinal fever, variola, septicemia, pneumonia, and the exanthemata.

(*b*) Profound *blood changes* such as occur in leukemia, scurvy, and the various cachexias.

(*c*) *Degenerations of the arterial walls*, such as occur in arterio-sclerosis, sometimes as the result of syphilis, nephritis, etc.

(*d*) The ingestion of *toxic substances*, such as quinine belladonna, ergot, mercury, the iodides, and the snake poisons.

(*e*) *Mechanical strains*, such as violent coughing and convulsions.

(*f*) *Neurotic conditions*, *e. g.*, vaso-motor relaxation due to hysteria, spinal cord inflammation, etc.

(*g*) *Hemophilia.*

Treatment of Purpuric Diseases.—In the secondary purpuras, treatment should be directed to the causal condition. In simple purpura, *arsenic* is of great value. In rheumatic purpura the most important remedies are *bryonia*, *arnica*, *ledum*, *mercurius*, and *belladonna;* and in hemorrhagic purpura *arsenic*, *lachesis*, *naja*, or *phosphorus* is usually indicated.

SCURVY.

(Scorbutus.)

Etiology.—This disease is the result of depraved vitality, due to:

1. Improper diet, *i. e.*, an insufficient supply of fresh vegetables.

2. Insanitary surroundings.

3. Infection. Analogy affords ground for a suspicion that the disease may be infectious.

Pathology.—1. Profound anemia of symptomatic type, with fatty degeneration of the viscera.

2. Hemorrhagic effusions into all tissues and into the joints.

Symptoms.—The disease is gradual in its onset, presenting:

1. *General anemic symptoms*, including pallor, emaciation, weakness, palpitation, and edema. Irregular fever may occur, and gastro-intestinal disturbances, especially diarrhea, may be prominent.

2. *Hemorrhagic symptoms.* The gums become swollen and tender and bleed readily. The teeth may be loosened or even drop out. Ecchymotic spots appear on the skin, especially about the ankles, and occasionally ulceration follows. Subperiosteal hemorrhages are common, especially in children, and may lead to pain and swelling in the affected limbs. Bleeding may arise from any of the mucous membranes, and constitutes a serious symptom.

Prognosis and Clinical Course.—Under suitable conditions the prognosis is favorable. The course of the disease is chronic, extending over weeks, and death may finally occur from asthenia, edema of the lungs, pneumonia, or diarrhea. Ankylosis may follow hemorrhage into a joint.

Treatment.—Fresh air, fresh vegetables and fruit juices suffice to cure the disease. Convalescence may be hastened by the use of *mercurius* or the *mineral acids* (hydrochloric, nitric, or sulphuric). Hemorrhagic symptoms require *arsenic*, *cinchona*, *crotalus*, *hamamelis*, *lachesis*, *phosphorus*, or *terebinth.*

INFANTILE SCURVY.

(Barlow's Disease.)

As the result of the exclusive use of condensed milk or other preserved foods, infants are prone to develop subperiosteal extravasations of blood, which cause thickening and tenderness in the shafts of the long bones. There may also be intermuscular hemorrhages, but as a rule the joints escape. The child becomes anemic and feverish, and the pain on motion causes it to lie motionless, with the legs drawn up, and to cry out when moved. Prompt recovery follows the administration of fresh milk, beef juice, and the juices of orange, lemon, pine apple, etc. The remedies mentioned above include those ordinarily required by children.

DISEASES OF THE DUCTLESS GLANDS.

ADDISON'S DISEASE.

Etiology.—The lesions of the adrenal bodies, as the result of which the physiological activity of these glands may be abolished, are as follows:

1. Tuberculosis, with fibro-caseous degeneration or calcification.

2. Degeneration, fatty or cystic.

3. Atrophy, simple or following chronic interstitial inflammation.

4. Malignant disease (carcinoma or sarcoma).

5. Hemorrhagic extravasation or embolism.

It is probable that these glands furnish an internal secretion essential to normal metabolism, failure of which is followed by the group of symptoms known as Addison's disease. Another theory, however, ascribes the disease to the abdominal sympathetic system and regards the pigmentation as a trophic phenomenon.

Symptoms.—1. *Pigmentation of the skin* (melasma), the color varying from a light yellow to brown or almost black. This bronzing is deeper on exposed portions of the body, often appears on the mucous membranes, and may be associated with whitish patches of leucoderma. The tint is never uniform all over the body, even in extreme cases. The patches shade off gradually and have no abrupt margins.

2. *Asthenia.* The patient suffers with languor and debility, the heart's action is feeble, the pulse is small in volume and low in tension, and a symptomatic anemia is present. Emaciation does not occur.

3. *Gastric irritability.* The patient suffers more or less with anorexia, nausea, and vomiting.

Diagnosis.—Pigmentation of the skin is not pathognomonic of Addison's disease; it is sometimes associated with tuberculosis of the peritoneum, abdominal neoplasms, pregnancy, and uterine and hepatic diseases, and the possibility of each of these should be excluded. *Jaundice*, unlike Addison's disease, colors the conjunctiva and the urine. *Argyria*, the pigmentation resulting from the long-continued ingestion of silver nitrate, can be distinguished by the history. The co-existing asthenia and gastric irritability are strong confirmatory signs of Addison's disease. It has been suggested that, since a majority of the cases are tubercular, the tuberculin test be applied to doubtful cases.

Prognosis.—The disease is invariably fatal. The duration varies from a few months to several years, death occurring, as a rule, from asthenia.

Treatment.—Conserve the patient's strength by keeping him at rest. The food should be easily assimilable and highly nourishing, and stimulants frequently become necessary. Nausea may be relieved by carbonated waters, koumiss, champagne, etc. *Arsenic* and *argentum nit.* are strikingly indicated in many cases and unquestionably serve to control the symptoms for which they are prescribed. *Calcarea ars.*, *iodine*, and *theridion* have also been recommended, and the administration of *suprarenal extract*, gr. iij–vi, t. i. d., may be tried.

GOITRE.

(Bronchocele; Derbyshire Neck; Struma.)

Etiology.—The exciting cause of this enlargement of the thyroid gland is unknown. Certain drinking waters, or rather some constituent of them, have been proven to produce goitre, and iron pyrites, copper pyrites, sulphate or carbonate of lime, and carbonate of magnesia are among the substances credited

with this action. In certain localities in Europe goitre is endemic, but in this country it is only sporadic.

Pathology.—According to their anatomical features, goitres are divided into the *parenchymatous*, a simple hypertrophy of the gland; the *fibrous*, in which the increase is chiefly of connective tissue; the *cystic*, in which the normal follicles become enlarged to form cysts with liquid contents; the *colloid*, in which the cysts contain a colloid material; the *calcareous*, in which calcareous infiltration occurs; and the *amyloid*, in which there is a wax-like product. Rarely the enlargement is associated with great dilatation of the vessels, whereby a forcible expansile pulsation is imparted to the gland—*pulsating goitre.*

Symptoms.—Subjective symptoms are usually lacking, the only characteristic indication of the disease being a swelling over the region of the thyroid which *moves with the larynx in deglutition.* By pressure the enlarged gland may cause dyspnea or dysphagia, or by interference with nerves, especially the pneumogastric, it may induce spasm of the glottis or paralysis of one or both vocal cords.

Treatment.—*Iodine*, used locally and internally, is the leading remedy. *Baryta iod.*, *bromine*, *calcarea carb.*, *kali iod.*, *phytolacca*, and *spongia* are also recommended. In obstinate cases an alcoholic solution of iodine may be injected, electrolysis may be attempted, or surgical extirpation of the gland may be resorted to. Patients should either remove from a goitrous district or drink only boiled water.

EXOPHTHALMIC GOITRE.

(Grave's or Basedow's Disease; Tachycardia Vasomotoria.)

Etiology.—Two theories as to the origin of exophthalmic goitre are in vogue, viz.:

1. That it is due to a lesion in the medulla or the sympathetic nervous system.

2. That it is the antithesis of myxedema, being due to overactivity of the thyroid gland.

Symptoms.—Four symptoms are conspicuous features of this disease, though one or more of them may be absent in a given case.

1. *Tachycardia.* The pulse rate is usually above 100, and may reach 200. The heart impulse is strong, the sounds are loud, and a soft blowing murmur is often heard at the base.

2. *Exophthalmos.* Both eyeballs may be protuberant, though sometimes one is more so than the other; and at times the protrusion is so great that the lids are unable to close. In some cases the lid does not not follow the eye when it is cast downward or raised (von Graefe's sign), or there may be increased width of the palpebral fissure, owing to the retraction of the upper lid (Stellwag's sign). The patient winks less frequently than is normal.

3. *Thyroid enlargement* can usually be discovered; it is moderate in degree, by no means as great as in a case of simple goitre. Pulsation and thrill may be detected on palpation, and auscultation reveals a blowing murmur.

4. *Tremor* is almost invariably present. It is very fine, becomes aggravated under any excitement, and is associated with vague "general nervousness."

Various other symptoms, such as menstrual disturbances, local or general edema, gastro-intestinal derangement, etc., may be noted in connection with some cases.

Diagnosis.—In the presence of tachycardia search should always be made for other evidences of exophthalmic goitre. The discovery of slight temporary enlargement of the thyroid or of tremor will justify a positive diagnosis. Without the presence of tachycardia, however, no case can be considered one of exophthalmic goitre.

Prognosis.—The disease is chronic in its course, usually persisting for several years. Under proper treatment a majority of the patients improve and many recover. Death may occur from heart failure or intercurrent disease.

Treatment.—Rest, preferably absolute for a time, and freedom from excitement, are essential preliminaries to successful treat-

ment. The diet should be highly nutritious, and daily massage and a morning cold sponge bath are advantageous. *Lycopus* is usually the most efficient medicine, but indications may exist for the administration of *arsenic*, *aurum*, *belladonna*, *ferrum*, *glonoin*, *iodine*, *sulphur*, and many other drugs.

MYXEDEMA.

Etiology.—As a result of some change in the thyroid gland which renders the latter functionless, a substance essential to general nutrition, probably a secretion, is no longer elaborated; and in consequence a solid edematous swelling of the subcutaneous tissues appears.

Symptoms.—The disease is much more common in women than men, and is apt to appear during middle life. The onset is insidious, the condition becoming noticeable only when the nutritional change is so advanced that the patient's *face is uniformly swollen* and flattened, the nose being broad, the eyelids puffed, and the usual wrinkles obliterated. The *skin is dry* and harsh, pale and waxy in appearance, and does not pit on pressure. The hands are swollen and "spade-like," and the feet are enlarged. The *mind is torpid*, mental processes are slow, and dementia may ensue. The temperature is subnormal.

CRETINISM is myxedema occurring in infancy or childhood, as the result of which there is an almost complete arrest of mental and physical development, and the patient becomes an idiot and a dwarf. The general symptoms do not differ from those of adult myxedema.

OPERATIVE MYXEDEMA (*cachexia strumipriva*) is myxedema following surgical extirpation of the thyroid gland.

Diagnosis.—The swollen appearance, with the absence of pitting on pressure, the harsh, dry skin, the slowness of thought, speech, and action, and the subnormal temperature, form a clinical picture which is unmistakable.

Prognosis.—Previous to the introduction of thyroid feeding the prognosis was hopeless, but under that method of treat-

ment the symptoms of myxedema can be entirely removed in a few months.

Treatment.—During the early stage of the treatment keep the patient at rest, and administer from 3 to 10 grains of the powdered extract, or from 15 to 30 minims of the glycerine extract of the thyroid gland, three times a day. Watch the effect carefully; acceleration of the pulse demands reduction in the dose, while a fall of temperature or a slight increase of the swelling requires an increase. As improvement begins the dose may be reduced slightly. After the myxedema has been removed a single small daily dose must be continued for an indefinite period.

CONSTITUTIONAL DISEASES.

DIABETES MELLITUS.

Etiology.—Among the causes predisposing to diabetes mellitus are heredity, male sex, and middle life. Intellectual workers, the better classes, and especially Hebrews, are among those most often affected. The obese are liable to the alimentary form, while a special variety is due to traumatism, especially injuries to the head.

Pathology.—Diabetes is in reality a generic term, for glycosuria may result from:

1. *Organic disease of the pancreas.* Extirpation of the pancreas is followed by glycosuria, and that organ is found diseased in a majority of diabetics; and these include the most severe cases.

2. *Interference with the glycogenic function of the liver.* This may result from disease of that organ or of the nervous system (puncture of the floor of the fourth ventricle, section of the pneumogastric nerve, etc.). These are the cases which follow central or spinal disease.

3. *Ingestion of an excess of carbohydrates.* If more carbohydrates are taken into the body than can be consumed in the tissues or stored in the liver as glycogen, the excess finds its way into the urine as sugar. This constitutes alimentary glycosuria.

Morbid Anatomy.—The post mortem findings are not distinctive. The most common is a granular atrophy or other degenerative change in the pancreas; rarely, organic disease (tumors, sclerosis, etc.) is found in the medulla, and yet more rarely there is primary disease of the liver. The proportion of sugar in the blood is increased, and the profound influence

of the disease on general nutrition is demonstrated by secondary changes in many organs, including the liver, kidneys, heart, lungs, brain, cord, and peripheral nerves. Peripheral neuritis is particularly common, and when multiple has been incorrectly termed diabetic tabes.

Symptoms.—An immense number of symptoms, none of them necessarily distinctive, may be grouped about the essential ones of glycosuria, polyuria, thirst, and wasting. They may be classed as:

1. *General Symptoms.*—Bodily and intellectual weakness comes on gradually, with increasing emaciation, distressing thirst, and insatiable hunger.

2. *Cutaneous Symptoms.*—The skin becomes dry and harsh, itching is often intolerable, the hair may fall out, and boils and carbuncles are common. Gangrene may supervene.

3. *Nervous Symptoms.*—Depression of spirits, headache, and an irritable temper are common in diabetics. Peripheral neuritis may occasion localized numbness, tingling, trophic disturbances, and loss of reflexes (*diabetic tabes*). Diabetic coma may furnish a terminal catastrophe.

4. *Urinary Phenomena.*—The urine is increased in quantity, and this increase is sometimes so great that it is remarked by the patient and brings him to the physician. It is pale in color, acid in reaction, its specific gravity is high (1020–1065), and it contains sugar in amounts varying from ½ to 10 per cent. Albumin is not unusual.

Clinical Varieties.—By the severity of the clinical manifestations two distinct forms of the disease may be recognized, which also presumably differ in their underlying cause, viz.:

1. *Pancreatic Diabetes.*—This is a severe variety in which dietetic restriction is unavailing, albuminoid disintegration being persistent, the patient becoming extremely emaciated and dying within a few years.

2. *Alimentary (Lipogenic) Glycosuria.*—This is a mild variety due to excessive eating and drinking and physical inactivity. It occurs, therefore, in the obese, in those beyond

middle life, and is frequently associated with gout. As a rule it can be controlled by proper dietetic restrictions.

Course.—Some cases of diabetes, especially in the young, are acute in onset and run a rapidly fatal course. They are probably of pancreatic origin. A majority of even the severe cases run a more or less protracted course extending over several years, death finally occurring from one of the complications or from coma. The alimentary form is compatible with a long life, the tendency being to the supervention of nephritis.

Complications.—Gangrene, tuberculosis, arterio-sclerosis, cardiac weakness, interstitial nephritis, cataract, optic neuritis.

Diagnosis.—By the discovery of persistent glycosuria.

Prognosis.—An onset late in life, in association with obesity or gout, slight glycosuria, tolerance for a certain quantity of carbohydrates, and circumstances which permit hygienic and dietetic restrictions render the prognosis for life quite favorable. On the other hand, youth, rapid loss of strength, grave complications, intense glycosuria, intolerance of even small amounts of carbohydrate food, and poverty, render the prospect unfavorable. In any case, imminence of coma and a fatal issue is indicated by the appearance of diacetic acid in the urine. (For test, see page 201.)

Treatment.—1. *Dietetic.* At once place the patient on a diet free from carbohydrates; it may include meats, soups, oysters, clams, fish, poultry, game, eggs, green vegetables, spinach, celery, salads, butter, cheese, tea, coffee, whisky, and mineral waters. These articles must be prepared without sugar or dressings which contain flour. Saccharine may be used to sweeten the tea or coffee. Following the adoption of this diet, examine the urine daily for a week. By the end of that time sugar will have disappeared in mild cases and will be much diminished in those of moderate severity. Then allow the patient a definite, weighed quantity of carbohydrates (wheaten bread or baked potato) daily. If the first quantity, say 4 ounces, does not cause a reappearance of the sugar, or an increase over the previous minimum, cautiously increase

the amount of carbohydrate until the glycosuria does reappear or increase. Then drop back to the largest amount of carbohydrate food which was tolerated without increase of sugar, which may be considered the patient's maximum allowance.

2. *Hygienic.* The patient must live in the open air as much as possible, must exercise moderately but persistently, and he should take a cool sponge bath on arising in the morning, and a tepid bath at least twice a week on retiring.

3. *Medicinal.* Indications may be found for many remedies. *Uranium nitrate* is particularly indicated in severe, presumably pancreatic, cases, and its effects are sometimes striking. *Phosphoric acid* is advisable for over-worked, weak, neurotic patients who pass large quantities of highly saccharine urine, rich in phosphates or oxalates. *Phosphorus* may be preferred for older persons with degenerated tissues. *Arsenic* or *sodium arsenite* is useful in cases where anemia, gastro-intestinal disturbances, and cutaneous symptoms are prominent. *Aurum* is suggested by cardiac weakness associated with neurotic symptoms. A complicating nephritis may call for the administration of *aurum*, *plumbum*, or *plumbum iodide*. Special symptoms will afford indications for other remedies.

DIABETES INSIPIDUS.

Etiology.—This disease is marked by prolonged excretion of an excessive quantity of non-saccharine and non-albuminous urine. Its cause is unknown, but a large proportion of cases is associated with cerebral disease or injury, and inasmuch as puncture of the floor of the fourth ventricle above the diabetic centre produces polyuria, it seems probable that the lesion lies in the nervous system.

Symptoms.—The principal symptom is *increased secretion of urine*, the quantity varying from six to forty pints in twenty-four hours. The urine is pale, often clear as spring water, and of low specific gravity. In addition the patient complains of *intense thirst*, and there is *dryness of the skin* and absence of

perspiration. The appetite is voracious, but the patient becomes progressively feeble and emaciated, and complains of vague nervous symptoms. The course of the disease varies greatly; the patient may die from exhaustion within twelve months, or he may live for years and finally die of some intercurrent affection.

Diagnosis.—*Simple polyuria*, the result of nervous conditions, diuresis, or excessive imbibition of fluids, is readily distinguished by its temporary character.

Interstitial nephritis may for a time present a large amount of non-albuminous urine, but persistent re-examination will finally reveal albumin and tube casts, which, in association with cardio-vascular changes, permit of no diagnostic doubt.

Prognosis.—Recovery is not infrequent, and death occurs, as a rule, only in cases with symptoms indicating a serious nerve lesion. Many cases are protracted but "troublesome rather than dangerous."

Treatment.—No limitation of the amount of fluids consumed is advisable. Medicines should be prescribed in accordance with the symptomatic indications, the more important being *argentum*, *aurum*, *murex*, *phorphoric acid*, *scilla*, *strophanthus*, *uranium*, and *valerian*.

GOUT.

Etiology.—An abnormal accumulation of uric acid in the blood and tissues may be the result of:

1. Heredity (60%).
2. Excessive indulgence in rich food and liquors.
3. An inactive life.

The disease occurs more frequently in males and in middle or advanced life. Lead-poisoning is a contributing cause in some cases.

Pathology.—As the result of decreased elimination or increased formation an excess of uric acid is found in the blood, and in the form of sodium urate it may be deposited around

the fibrous structures of the small joints, especially the great toe. The deposit may be uniform throughout the tissue, or it may appear in small opaque areas, forming "chalk stones" (tophi). The latter appear upon the digital joints, the cartilages of the ear, and less often about the vocal cords, the cartilages of the nose, etc. Those about the joints sometimes ulcerate through the skin. Ultimately sclerotic changes are induced in the arterial system, the kidneys, and other organs.

Varieties.—1. Regular gout (acute or chronic).

2. Irregular gout (lithemia; uric acid diathesis).

Symptoms.—*Acute gout.* Often after symptoms of indigestion or some similar disorder the patient is seized with *agonizing pain* in one of his joints, usually the metatarso-phalangeal joint of the great toe, about which there is swelling, heat, and discoloration of the skin. There is moderate fever (100–102°), and the *urine is scanty*, dark-colored, of high specific gravity, and contains an excess of uric acid and urates. In the course of some days the symptoms subside gradually, the joints become more supple, the skin over the affected articulation desquamates, and convalescence ensues.

Gout is said to be *retrocedent* when it suddenly disappears from the joints and occasions derangement of some internal organ, such as the brain, heart, stomach, or bladder. Such cases may properly be regarded as forms of irregular gout, however.

Chronic gout represents the gradual accumulation of morbid changes induced by repeated acute attacks. The joints become filled with chalky deposits, immobility and deformity ensue, and finally cardio-vascular degeneration and chronic interstitial nephritis become associated with the arthritic conditions.

Irregular gout includes a variety of symptoms which are not in themselves distinctive, but which represent a condition of disturbed metabolism with an excess of uric acid in the blood. It lacks the uratic deposits in the tissues and the other localized manifestations of regular gout, and instead presents

a wide variety of *nervous symptoms* (headaches, neuralgias, insomnia, lumbago, etc.), *digestive disturbances* (coated tongue, dyspeptic symptoms, gastralgia, hyperchlorhydria, and constipation), and various *cutaneous irritations* (pruritus, eczema, etc.), together with circulatory and respiratory disorders. In this, as in the regular form, the diagnosis rests upon the urinary phenomena.

Diagnosis.—Regular gout may be mistaken for *rheumatism*, but the latter usually affects larger joints, such as the knee and elbow, presents higher fever and profuse acid sweats, and desquamation of the epidermis rarely follows the attack.

Irregular gout may simulate various nervous and gastro-intestinal affections, and often is passed over as "malaria" or "rheumatism;" but the acid, high-colored, scanty urine, loaded with uric acid and urates, while not in itself absolutely diagnostic, becomes conclusive evidence when associated with the history and symptoms detailed.

Prognosis.—In acute gout and in irregular gout, treatment is usually effective. In chronic gout, and when secondary changes have occurred in heart, arteries, and kidneys, the outlook is much less favorable.

Treatment.—1. *Diet.* Meats, alcohol, tea, coffee, and tobacco must be restricted, while milk, butter, fruits, and succulent vegetables are recommended. A moderate amount of fish, oysters, and poultry may be substituted for butcher's meat.

2. *Hygiene.* The patient should take a morning cold bath or an evening warm bath daily, the bowels and skin should be kept active, and he should exercise regularly in the open air.

3. *Medicines.* During an acute attack the patient should remain in bed with the affected joints wrapped in cotton, and the administration of *colchicum* or *colchicine* is generally indicated. In some cases *aconite*, *arnica*, *belladonna*, *benzoic acid*, or the *benzoates*, *berberis*, or *bryonia* may be preferable. In the periods between attacks or in cases of irregular gout a great variety of remedies, especially *berberis*, *lycopodium*,

sepia, and *sulphur*, may be symptomatically indicated; but it must be remembered that cure depends almost entirely upon proper diet and habits of living.

ARTHRITIS DEFORMANS.

(Rheumatoid Arthritis; Rheumatic Gout; Osteoarthritis.)

Etiology.—This deforming disease of the joints occurs independently of rheumatism or gout and their causes. It attacks women much more frequently than men, and the more important predisposing causes are heredity, traumatism, insufficient food, and such neurotic factors as shock and worry.

Pathology.—The exact nature of the process is undecided, many regarding it as of neurotrophic origin. and others considering it but a manifestation of the arthritic diathesis. The joint changes consist of proliferation of the cartilage cells, followed by fibrillation of the intercellular substance and subsequent degeneration, liquefaction, and absorption, which leaves the bone ends bare; the latter finally become smooth and eburnated. The remaining peripheral margin of cartilage thickens and forms bony outgrowths (*osteophytes*), the synovial membrane becomes thickened and its fringes hypertrophied, and as a result the joint is stiffened and creaks or grates when moved, the muscles about it undergo atrophy, and great deformity ensues.

Symptoms.—The onset of the disease is very insidious. The patient, often a youngish woman, notices a little tenderness or swelling in a joint, and soon one or more articulations become painful, swollen, and inclined to creak on motion. Nodules develop on the sides and ends of the distal phalanges of the fingers and sometimes the toes (Heberden's nodosities, seen also in gout). The ligaments about the joints become thickened, and muscular contractures flex the limb in an abnormal position. The disease advances slowly, often by a series of acute exacerbations which attack fresh articulations. There is a tendency to involve corresponding joints on the

two sides, the disease progressively invading the small joints of the hands, the knees, the feet, ankles, wrists, elbows, shoulders, hip joints, and even the spinal column. The joints become less and less mobile and undergo flexion and contracture, so that finally the fingers are bent backward and turned toward the ulnar side, the thighs are drawn up, the legs adducted and flexed, and the condition of the motionless and distorted patient is truly pitiable. She becomes emaciated and anemic, suffers much pain at times, and yet lives for years.

Varieties.—*Multiple* arthritis deformans, in which the large joints are progressively invaded.

A *partial* or *monoplegic* form, limited to one or two joints, which is particularly common in the hip-joint of elderly men and is associated with wasting of the muscles of the buttock and the thigh.

Diagnosis.—*Gout* is distinguished by tophaceous deposits and an excess of uric acid in the blood and urine. *Rheumatism* is accompanied by a fever and a tendency to heart complications. The arthropathies of *locomotor ataxia* and *syringomyelia* are distinguished by the associated symptoms and the absence of osteophytes.

Prognosis.—Generally unfavorable, though spontaneous arrest may occur. The duration is protracted, death occurring only as the result of intercurrent disease.

Treatment.—Improve nutrition by the use of such foods as cream, butter, fat meat, and cod liver oil. Maintain joint movement by active and passive movements and massage. Steam or electric baths and local galvanism may be tried. Of medicines, *calcarea*, *cimicifuga*, *pulsatilla*, *sabina*, and *sulphur* are especially recommended for cases occurring in women; *aurum*, *benzoin*, *causticum*, and many other drugs are symptomatically indicated at times. Acute exacerbations are benefited by *colchicine*. *Ferrum iodide* may be tried in the form of the syrup.

RICKETS.

(Rachitis.)

Etiology.—This nutritional disease of childhood is due to improper feeding and insanitary surroundings, and in consequence it is found principally in the children of the tenements in large cities.

Pathology.—The characteristic lesion is an increased production of cartilage at the epiphyses and excessive cell-growth beneath the periosteum, with slow or arrested ossification, whereby the bones become unnaturally flexible and deformed. There is a decided deficiency of lime salts in the bones. The liver and spleen are enlarged, and catarrhal inflammations of the respiratory and gastro-intestinal tracts are common.

Symptoms.—In a child, usually less than two years of age, mild symptoms of ill-health, such as restless sleep, digestive and nervous disturbances, and excessive sweating about the head, may attract attention. Delayed dentition and muscular weakness may also be noticed. On examination the characteristic "rickety" condition is found; the head is large in proportion to the face, or the ribs are beaded (rachitic rosary), the knees are bent or bowed, the spine curved, the belly prominent, and the skin pale. Convulsions, tetany, and laryngismus stridulus are not uncommon occurrences in these children.

Complications.—The rickety child is weak and therefore peculiarly susceptible to all the ills of childhood, especially bronchitis, broncho-pneumonia, pulmonary collapse, and diarrheal troubles.

Prognosis.—The disease in itself is not fatal, and if complications can be avoided the course, though prolonged over months, tends toward recovery. The deformities are, however, persistent.

Treatment.—See that the child is properly housed, fed, bathed, aired, etc., and prescribe such medicines as *phosphorus*,

calcarea phos., *calcarea carb.*, *ferrum phos.*, *phosphoric acid*, *silica*, etc. Orthopedic apparatus or even surgical operation may be necessary to straighten the distorted bones.

OSTEOMALACIA.

(Mollities Ossium; Malacosteon.)

Etiology.—The causes of this rare affection are little understood. It occurs principally in women, especially in connection with pregnancy and lactation; a smaller proportion of cases may be attributed to starvation, and a few to senility. The disease appears to be common in certain geographical locations.

Pathology.—The calcium salts of the bones are absorbed by the fluids of the body, being found in increased proportion in the blood and excretions; and as a consequence the bones become softened and distorted, the pelvis in particular being deformed and narrow.

Symptoms.—The onset is attended with pain of a rheumatoid type in the portion of the body first attacked, and after a time deformity becomes apparent, the bones being twisted or bent. Fractures may result from slight traumatism, and, refusing to reunite, may form false joints.

Prognosis.—Recovery is rare, although the disease pursues a chronic course and death may be deferred for five or ten years. Temporary arrests may occur.

Treatment.—Attention should be directed to the patient's hygiene; she should have an abundance of good food, and pregnancy must be avoided. *Phosphorus* is the principal remedy, although the *calcareas* and other nutritional remedies should be considered.

OBESITY.

(Polysarcia; Adipositas Universalis.)

Etiology.—The abnormal accumulation of fat throughout the body appears to be the result of impaired oxidation. Factors

favoring it are heredity, over-eating of either carbohydrates, fats, or proteids, and muscular inactivity. Anemia and chlorosis favor the accumulation of fat through defective oxidation, and sexual continence also contributes to it.

Symptoms.—The increased bulk of the patient, which is seen at a glance, renders his movements sluggish and his gait waddling. In time the fat interferes mechanically with the heart and with respiration, inducing dyspnea. Cardiac hypertrophy may ensue, but later fatty infiltration of the myocardium leads to failure of the heart, and arterio-sclerosis, interstitial nephritis, or diabetes mellitus may be added.

Prognosis.—While corpulence may not be incompatible with long life, it tends to produce the pathological conditions noted. Recovery is quite generally possible, however, provided the patient will persist in the somewhat irksome treatment.

Treatment.—1. Limit the ingestion of fat-producing food. Cut down the allowance of all food, but especially that of carbohydrates, and allow only a small quantity of fluid with meals.

2. Promote oxidation. Exercise, in the form of gymnastic work, walking or climbing, and bicycling, together with massage and Turkish or steam baths, accomplishes this purpose.

The administration of drugs is not advisable.

DISEASES OF THE NERVOUS SYSTEM.

Disease of the nervous system manifests itself by disorders of intellection, of sensation, of muscular action, of reflex action, or by vasomotor or trophic disturbances.

Disorder of the *intellectual functions* may occasion loss of memory, disturbed sleep, hallucinations or delusions, delirium or coma, and various abnormalities of speech. Disturbances of *sensation* may lead to diminution of sensibility (anesthesia), increase of sensibility (hyperesthesia), or abnormal sensations (paresthesia). The skin may be disordered as to the sense of touch, of locality, of position, of pressure, of temperature, or of pain. Abnormalities of *muscular action* are manifested in paralysis, in spasm or convulsion, or inco-ordination.

Disorder of the reflexes is rarely discovered until some of the symptoms already mentioned have led to their investigation. *Vasomotor* disturbances may be evidenced by abnormal redness or pallor of the skin; and *trophic* disorders lead to wasting of certain portions of the body.

Interrogation of the Patient.—Attention having been attracted by phenomena suggesting disease of the nervous system, certain questions concerning heredity, environment, habits, and previous disease become of extreme importance. The patient should be questioned as to:

1. His *family history*. Have there been mental diseases, chorea, convulsions, or paralysis?

2. His *personal history*. Has he ever suffered from rheumatism, gout, syphilis, or alcoholism?

3. His *work*. Is he exposed to noxious influences or poisons, such as lead, mercury, or arsenic?

4. His *environment.* His home surroundings, etc.

5. His *habits*, as to eating, sleeping, stimulants, venery, work, and recreation.

6. In *cerebral* cases: Has there been any discharge from the ear? Any injury to the head?

7. If there have been *convulsions*, inquire as to: His *age at the time of the first convulsion*, and the circumstances surrounding it. The supposed cause. Describe in detail the first attack. When did the second occur? What has been the longest and what the shortest interval between the attacks? Are they more, or less, frequent now? Do they occur during sleep? Has he any aura? If so, what and where? Is the onset sudden or gradual? Does he remain rigid, or does he struggle? Are the convulsive movements uni- or bilateral? Where do they begin? Does he bite his tongue, urinate, or defecate during the seizure? Has he ever hurt himself during the attack? How does it end? Are there any after-symptoms, such as sleep, headache, or automatism?

8. If there be *paralysis* ascertain its exact location. Did he have any premonitory symptoms before the onset? Did it develop abruptly or gradually? Was he unconscious at the time; and if so, for how long? Has he any headache? Where is it situated? Is it worse at night? Has he any giddiness? Any difficulty in walking? Inquire carefully for any symptoms of heart or kidney disease.

EXAMINATION OF THE NERVOUS SYSTEM.

The close association of nervous phenomena with every disease of the human body renders it absolutely essential that knowledge of the condition of each important organ, as well as whatever information may be derived from investigation of the various excretions, shall be in the possession of the physician before he formulates an opinion as to the case. Examination of the thoracic and abdominal viscera, and of the urine, stomach-contents, or blood, should, therefore, in many

cases precede the special examination directed to the nervous system. By means of the latter he must, moreover, determine two points:

(1) The nature of the lesion; and

(2) Its localization. For this purpose a certain degree of familiarity with the anatomy and physiology of the nervous system is necessary.

I. THE INTELLECTUAL FUNCTIONS.

As a rule the *mental condition* can be judged with fair accuracy while obtaining the history of the case. Note, in the first place, whether the patient is *right* or *left-handed.* Test his *memory* by asking him what day of the week it is, or what he had for breakfast. Inquire as to how he sleeps. Note his *emotional condition*, whether excited, irritable, or inclined to burst into tears. Ascertain whether any hallucination or delusion is entertained. The presence of delirium or stupor will, of course, prevent this investigation.

Notice his *speech.* Does he stammer? Does he speak deliberately, as though *scanning* a line of poetry (disseminated sclerosis); or does he slur his words like a drunken man (paresis)? These are disorders of articulation (dysarthria, anarthria) and may be due to lesions of the muscles, their nerves, or of the latter's nuclei of origin.

If it is not articulation but the ability to speak at all, or else to understand speech when written or spoken, that is disordered, the condition is due to a cerebral lesion and is known as *aphasia.* The cerebral speech mechanism consists of:

(1) Two receiving portions, one for written and one for spoken speech; and,

(2) Two producing portions, one for speaking and the other for writing.

Failure of the receiving portion (*sensory aphasia*) may, therefore, be visual (*word blindness*) or auditory (*word deafness*); and failure of the producing mechanism is evidenced

by *motor aphasia*, with inability to talk (*aphemia*), inability to write (*agraphia*), or inability to use the sign language (*amimia*). Two or more of these conditions are associated in most cases.

Localization.—The site of the lesion producing motor aphasia is about the third frontal convolution (Broca's); that of sensory aphasia is in the neighborhood of the first frontal convolution. In right-handed subjects the lesion is on the left side of the brain; in the left-handed it may be on the right side.

The *lesions* producing aphasia are of many kinds, among the more important being intracranial tumor, abscess, gumma, thrombosis, embolism, softening, and depressed fracture of the skull. Occasionally it is without organic cause, being due to hysteria, epilepsy, migraine, or other neurosis.

Tests. Having found the patient to be in some degree incapable of voluntary utterance, discover whether his hearing is good. If it is, ask him to put out his tongue or close his eyes, and to whistle or to smile. If he does not do these things, there is *word-deafness*.

Show him some common object, such as a pen or a book, and ask him to name it. If he cannot, he forgets some words (*amnesic aphasia*). If he applies the wrong name, calling the pen a book or *vice versa*, he has *paraphasia*.

Ascertain if his sight is good. If it is, write on a piece of paper some simple question, such as "How old are you?" If he does not reply satisfactorily, there is *word-blindness*.

Ask him to write his name (if he has word-deafness, put the question in writing), and if he does so, test him further with some simple question. If he writes a satisfactory reply, there is no *agraphia*. Ask him to write a short account of his illness and note whether he uses the wrong word at times (*paragraphia*).

Ask him to copy a simple sentence from a book. If he does so, ascertain if possible whether he understands its meaning. If he does not, he has *alexia*.

Note whether he nods his head for "yes" and shakes it for "no." Loss of this sign language is termed *amimia;* or if he misuses the gesture, shaking his head when he means "yes," there is *paramimia.*

II. CRANIAL NERVE FUNCTIONS.

1. First or olfactory nerve. Apply to each nostril separately two small bottles containing respectively oil of peppermint and tincture of asafœtida, and ask the patient if he recognizes the difference. There may be a loss of the sense of smell (*anosmia*), increased sensitiveness of smell (*hyperosmia*), perversion of the sense of smell, in which, for example, offensive odors seem pleasant (*parosmia*), and there may be *hallucinatiens of smell* (occurring sometimes as an epileptic aura).

2. Second or optic nerve. (*a*) Test the visual acuity of the patient by means of the ordinary Snellen types; if, for instance, at a distance of 10 feet he is able to read only the type normally visible at 20 feet, his vision is recorded as $\frac{10}{20}$; if he is able to read that normally visible at 15 feet, his vision is $\frac{10}{15}$, and so on.

If acuity of vision is much diminished, place him with his back to the light and hold up in front of him different numbers of fingers, asking him how many he sees. If there is a question as to whether he can distinguish light from darkness, test him by flashing light into his eyes with a head-mirror, asking him to say when it is dark and when light.

There may be defective vision without change in the fundus (*amblyopia*), or defective vision in one eye due to a lesion in the opposite side of the brain (*crossed amblyopia*), and there may be complete blindness without ocular change (*amaurosis*).

(*b*) Test the *field of vision* by sitting in front of the patient, having him place his hand over one eye and keep the other eye fixed on the examiner's face; and then bring some object, such as a pencil or a finger, gradually into his field of vision,

first from one side, then the other, then from above, and finally from below. More accurate results may be had by using the perimeter.

The field of vision may be contracted all around the periphery (*concentric contraction*) as in hysteria, optic atrophy, and retinal disease. Vision may be lost in the centre of the field (*central scotoma*) as the result of retrobulbar neuritis, which is usually bilateral, or because of disease of the choroid or retina, when it may be unilateral. Finally, there may be loss of sight in one-half of the field of vision in both eyes (*hemianopsia*); this may affect the temporal half of each field (*temporal hemianopsia*), it may affect the nasal halves (*nasal hemianopsia*) or it may affect corresponding sides of each eye (*homonymous hemianopsia*).

The color field may be tested in a somewhat similar fashion, using blue, yellow, red, and green cards. Normally the blue field is largest, the others following in the order named. Concentric contraction of the color field occurs in *hysterical amblyopia*. *Color blindness* invalidates the test; to discover it, let the patient attempt to match skeins of wool of different colors in good daylight.

3. The motor nerves of the eye (third, fourth, and sixth) should be tested together. A lesion of any one of them may produce (1) strabismus, (2) defective power of ocular movement, (3) diplopia. Inability to move the eye outwards, with diplopia on looking in that direction and possibly internal strabismus, indicates *paralysis of the external rectus* muscle (*sixth nerve*). If downward movement is impaired, with diplopia on looking down and possibly convergent strabismus, there is *paralysis of the superior oblique* (*fourth nerve*). If there is utter loss of movement except outwards and a little downwards, with ptosis, immobile pupil, and loss of accommodation, there is complete *paralysis of the third nerve*. The paralysis may, however, be partial, only one or two of the functions being abolished.

Tests (*a*). Place the patient with his back to the light and,

standing in front of him, direct him to follow with his eyes the movements of one of the examiner's fingers. Notice whether this action develops a squint in either eye; and repeat the test on each eye separately, noting its motility in every direction. Notice whether a squint is constantly present, even when the patient looks straight ahead, and whether it follows the sound eye equally in all its movements; if so, it is a *concomitant (spasmodic) strabismus*. If, on the other hand, the affected eye fails to follow the sound one when the patient attempts to turn it in the direction of action of the paralyzed muscle, it is a *paralytic strabismus*. Diplopia is usually present in the latter form only.

(*b*) *Defective power of convergence* of the eyes is ascertained by holding the finger about 18 inches in front of the patient's nose, telling him to keep looking at it. Gradually bring it nearer to his nose, and notice whether the eyes remain directed towards each other or whether they tend to diverge after first converging.

(*c*) *Diplopia* occurs in recent strabismus, but when the latter is of long duration the patient learns to disregard the false image and takes cognizance of only the true. In these chronic cases the false image may be restored by placing a piece of colored glass before one eye, preferably the sound one, when the false image will be seen at one side of the true one. Let the patient alternately close each eye; if the image to the right be that seen with the right eye, the diplopia is simple (*direct* or *homonymous*), but if the image to the right is seen with the left eye there is *crossed* diplopia.

Diplopia not dependent on eye disease is usually significant of serious nervous diseases (meningitis, tumor, abscess, disseminated sclerosis, syphilis). It may occur transiently, however, in alcoholic intoxication or cerebral concussion.

(*d*) *Abnormal ocular movements*. Ask the patient to look straight ahead, and then observe whether the eyes remain steady. Tell him to look to the extreme right, then to the extreme left, then upwards, then downwards; if rhythmical

oscillations of the eyeball are detected, there is *nystagmus*. The presence of this symptom is proof positive of organic disease (disseminated sclerosis, intracranial tumor, meningitis, etc.).

Note if the head and eyes are kept persistently turned to one side (*conjugate deviation*). If this be due to paralysis and the lesion be in the cerebral hemisphere, the eyes turn towards the side of the lesion. If, however, the lesion be irritative rather than paralytic, the deviation is towards the healthy side. If the lesion be in the pons, these conditions are exactly reversed.

Examination of the pupils. (*a*) Compare the size of the two pupils, first in a bright light and then in a dim one. Note whether they are small or large, and whether irregular in outline. *Irregularity* of the shape of the pupil may be due to adhesion of the iris to the lens, or perhaps be an early symptom of paralytic dementia.

(*b*) Mobility. Place the patient so that he faces a bright light, and cover each eye with a hand for half a minute. Withdraw one hand suddenly and watch the pupil; it should contract almost immediately and then after slight oscillation settle down to its normal size (*reaction to light*). Test each eye separately in this fashion.

Hold one finger near the patient's nose while he looks at a distant object, then suddenly tell him to look at the finger. As he does so, the pupils become much smaller (*reaction to accommodation*).

The pupil may react to accommodation but not to light (*Argyll-Robertson pupil*), indicating a lesion interrupting the reflex path between the optic nerve and the nucleus of the third nerve (locomotor ataxia, paretic dementia, etc.). If the pupil reacts neither to accommodation nor light, there is probably a lesion of the pupillary centre or the third nerve.

Fifth Nerve.—The trigeminus is a mixed nerve which supplies by its motor trunk the masticatory muscles, and by its sensory portion the skin of the face, the mucous membrane

of the mouth and nasal cavity, the conjunctiva, the cornea, and the anterior tongue with gustatory fibres (via chorda tympani), and the nose with olfactory fibres. It may be affected by lesions of the pons (hemorrhage, sclerosis) by injury or disease at the base of the skull, or by pressure upon or inflammation of its opthalmic, superior maxillary, or inferior maxillary divisions. The sensory portion may also be affected in hysteria, and by lesions of the posterior portion of the internal capsule; and the gustatory fibres may be influenced by facial lesions involving the chorda tympani.

Paralysis of the nerve, if complete, occasions loss of sensation in the areas of the skin and mucous membranes mentioned, together with difficulty in chewing. Trophic lesions may affect the cornea, and the salivary, buccal, and lachrymal secretions may be diminished. Spasm of the muscles of mastication (*trismus*) occurs in tetanus and other diseases; clonic spasm is exemplified in "chattering teeth." Irritation of the nerve may occasion facial neuralgia, and at times pain may be due to actual neuritis. This is frequently the case when inflammatory processes affect the orbit, antrum, or jaws.

Tests.—Motor functions. (*a*) Place the hands first on the temporal and then on the masseter muscles while the patient clenches his teeth. They should stand out with equal prominence on the two sides, but this will not occur on a paralytic side. There may be deviation of the jaw toward the paralyzed side on opening the mouth.

(*b*) *Sensory functions.* The common sensibility of the area supplied by the nerve is tested in the usual way. (See examination of the Sensory Functions.) Examine also the sense of taste. Use the following solutions: Sodium chloride (10%); tartaric acid (10%); strychnine sulphate (1–100); saccharine (1–250).

Ask the patient to put out his tongue and keep it out until the test is ended. Place the saccharine solution on the tongue, asking, "Is it sour?" If the taste is normal he will shake his head. Proceed in the same way with the other solutions, ask-

ing him, for instance, if the tartaric acid is sweet or bitter, etc.

A weak galvanic current may be used, as it occasions a metallic taste under normal conditions.

SEVENTH NERVE. The facial is the motor nerve of the face; it may be paralyzed (Bell's palsy) as the result of lesions in its nucleus, its cortical centre, or its trunk. As a result the face is drawn to the sound side, and the paralyzed side is smoothed of all its wrinkles, the eye cannot be completely closed, and the patient is unable to whistle.

Tests. (*a*) Ask the patient to close his eye as tightly as possible. Note that the upper lid droops as though heavy, while the eye is turned upward and quite a space remains uncovered Try to open the eye with the fingers while the patient endeavors to keep it closed; with the healthy eyelid this is very difficult, but when paralysis is present the eye may be opened quite easily.

(*b*) Ask the patient to whistle. He cannot.

(*c*) Ask him to smile. The mouth becomes drawn only to the healthy side.

Localization of the Lesion. Cerebral or supra-nuclear paralysis effects chiefly the lower part of the face. In peripheral or infra-nuclear paralysis the upper and lower portions are equally involved. A lesion of the nerve before it enters the aqueduct causes paralysis of the stapedius muscle, whereby the patient becomes extremely sensitive to loud sounds. A lesion within the aqueduct, except its outer end, involves the fibres of the chorda tympani and produces a paralysis of taste sensation in the anterior two-thirds of the tongue. It is necessary, therefore, to inquire as to the patient's sensitiveness to loud sounds, and to test his sense of taste as described in connection with the fifth nerve.

Spasmodic movements of the muscles supplied by the seventh nerve (facial spasm, convulsive tic) may include all the muscles, or only certain groups (*e. g.*, blepharospasm).

EIGHTH NERVE. The auditory nerve consists of two sets of

fibres, one of which supplies the cochlea, subserving the hearing, and the other supplies the vestibule and semi-circular canals, having to do with equilibration. The nerve may be affected anywhere in its course; in the cortical auditory centre (in the first temporo-sphenoidal convolution), in the nucleus about the floor of the fourth ventricle, in its course at the base of the brain, within the internal auditory meatus, or in its terminal filaments.

a. The sense of hearing may be (1) lost (*nerve deafness*); (2) hyperacute, so that even slight sounds are heard with painful intensity (*auditory hyperesthesia*, *hyperacusis*); this occurs in hysteria and in lesions of the facial nerve above the aqueduct, or (3) accompanied by subjective auditory sensations, such as "ringing in the ears" (*tinnitus*).

Tests.—(*a*) Exclude the presence of wax in the ear. Stand behind the patient and holding a watch at some distance from his ear, outside of the normal hearing range, bring it gradually nearer and ask him to speak when he hears it tick. Test each ear separately, the other being closed. It is necessary, of course, to know at what distance the watch used is heard by a healthy ear.

(*b*) If hearing is defective, decide whether it is due to a lesion in the nerve or in the middle ear. In order to do this, strike a tuning-fork and place its end against the middle of the patient's forehead. If the deafness is due to disease of the middle ear the patient will hear the tuning-fork louder on that side than on the healthy one, but if the deafness is due to auditory nerve disease the sound will be heard only on the healthy side. Deafness is probably due to nerve disease if hearing is better in a quiet place, if conversation is heard better than a watch, or if inflation of the middle ear renders the hearing worse.

b. Equilibration may be disordered, with resulting vertigo. In true vertigo external objects seem to move around the patient. The association of vertigo with deafness and tinnitus aurium constitutes the disease known as *labyrinthine vertigo* or *Meniere's disease*.

It must be remembered that vertigo may also be the result of disease elsewhere in the auditory apparatus, to an ocular palsy, to loss of muscular sensibility in the lower extremities, and to cardiac, gastric, and renal diseases.

NINTH NERVE. The glosso-pharyngeal supplies sensibility to the posterior third of the tongue and to the mucous membrane of the pharynx; motion to the stylo-pharyngeus and the middle constrictor of the pharynx; and the sense of taste to the posterior part of the tongue. This nerve has numerous communications, especially with the tri-facial, the facial, and the pneumo gastric nerves, and is rarely paralyzed alone. Symptoms of such a paralysis would be loss or perversion of the sense of taste and difficult deglutition.

Tests.—(*a*) Examine the sense of taste in the posterior part of the tongue.

(*b*) Tickle the back of the pharynx and notice whether the reflex is present.

TENTH NERVE. The pneumogastric or vagus is a mixed nerve of motion and sensation, some of its most important motor fibres being derived from the spinal accessory. The nerve is distributed to the pharynx, larynx, esophagus, trachea, lungs, heart, stomach, intestines, and spleen. It may be affected by lesions in the nucleus or in the nerve trunk.

Tests.—1. (*a*) *The pharynx.* Ascertain whether the patient has difficulty in swallowing. If paralysis is present, he usually complains of regurgitation of food through his nose or entrance of food into the larynx.

(*b*) Ask him to pronounce words which require complete closure of the naso-pharynx. If the paralysis is bilateral he is unable to do so, and "egg" becomes "eng" and "rub" becomes "rum."

(*c*) Examine the pharynx, using a tongue depressor, and note whether both sides of the palate arch upwards when the patient says "ah."

2. *The larynx.* Examine with the laryngoscope. Tell the patient to take a deep breath, and note whether the vocal

chords become more widely separated, as they should in health. If, instead, they come more closely together during inspiration, there is *total abductor palsy*, often due to central disease; or one cord may be normal, but the other lies near the middle line and does not move during inspiration—*unilateral abductor palsy*.

If the cords are normal in position and move normally in respiration, but are not brought together by the attempt at phonation, there is *adductor paralysis*. One cord may be moderately abducted and motionless while the other moves freely and even beyond the middle line in phonation—*total unilateral palsy*. Or both cords may remain moderately abducted and motionless—*total bilateral palsy*.

Paralysis of the vocal cords may be peripheral in origin, due to pressure on the recurrent laryngeal (by goitre, aneurism, tumors of the mediastinum, or pericarditis) or to neuritis; it may be central, as in hysteria; or nuclear, due to involvement of the nucleus of the accessory nerve in the floor of the fourth ventricle (bulbar paralysis, disseminated sclerosis, locomotor ataxia, syringomyelia, etc.).

It must be remembered that the laryngeal motor fibres of the pneumogastric are all derived from the spinal accessory, and that lesions of the latter before its junction with the pneumogastric may occasion laryngeal paralysis.

Eleventh nerve. The spinal accessory nerve is purely motor. One portion of it supplies the laryngeal muscles through the pneumogastric, as has been described. The other portion of it is distributed to the sterno-cleido-mastoid and trapezius muscles, which may be paralyzed by lesions of that portion.

Tests.—1. Paralysis of one sterno-mastoid occasions difficulty in rotating the head to the opposite side.

2. Paralysis of the trapezius may be detected by pressing upon the patient's shoulders while he tries to shrug them.

Twelfth nerve. The hypoglossal is the motor nerve of the tongue, and also supplies the depressors of the hyoid bone

It arises beside the olivary body in the medulla, and may be involved in disease of the cortex (cerebral hemorrhage, thrombosis, embolism, etc.), or of the nucleus (bulbar palsy, locomotor ataxia), or its fibres may be damaged in their course to the periphery.

Test.—Ask the patient to protrude his tongue as far as possible. Hypoglossal paralysis is indicated when the tongue, instead of being put out straight, deviates toward the paralyzed side. The apparent deviation of the tongue which appears when one side of the face is paralyzed must be distinguished from the true glossal paralysis just described.

In nuclear disease the palsy is apt to be bi-lateral, and in consequence the tongue lies motionless in the floor of the mouth. Note whether the tongue is atrophied; the latter condition indicates a nuclear or infra-nuclear lesion. Note if there be any tremor or spasm of the tongue.

III. MOTOR FUNCTIONS.

Examination of the motor functions implies investigation as to:

1. The existence of muscular paralysis or weakness.
2. The state of muscular nutrition.
3. The co-ordination of muscular action.
4. The occurrence of abnormal muscular movements.

1. **Muscular power.** Note the patient's ability to perform the ordinary muscular movements, such as walking or sitting up in bed. Note whether he can move each of his extremities. Having in this way detected any gross defects of motility, examination should be directed to the principal muscles and groups of muscles separately. The patient is asked to throw into action the muscle or muscle-group which is undergoing investigation, and while he does so the examiner interposes a certain degree of passive resistance to the action. If by this test a muscle or group of muscles is found to be feeble, the condition is described as a *paresis;* if voluntary motion is lost,

it is said to be a *paralysis*. When the presence of a motor defect has been determined, its degree and distribution must be ascertained; and for this purpose each movement—abduction, adduction, extension, and flexion—should be tested separately on the two sides of the body and compared, due allowance being made for the superior strength usually found on the right side. If the patient is unconscious, raise each limb separately and allow it to fall upon the bed; as a rule, the paralyzed limb drops limp and helpless, while the unaffected one offers slight resistance. It may be found that the paralysis is confined to the muscles supplied by a single nerve, *e. g.*, the musculo-spiral, or to a spinal segment, or it may constitute a hemiplegia, paraplegia, or monoplegia.

The power of individual muscles may be tested as follows (Butler), their innervation and their representation in the spinal segments being indicated in parenthesis:

(1) **Neck Muscles.** Practically the sterno-cleido-mastoid alone is examined. If the patient is lying down, request him to raise his head; or if he is sitting up, to turn the head as far as possible to the right and left, with or without resistance offered by applying the hands to the sides of the head; or to bend it forward against pressure on the forehead. If the muscle is not paralyzed it stands out prominently. (Spinal accessory nerve; medulla and second and third cervical segments.)

(2) **Muscles of the Upper Extremity.** *Deltoid.*—Request the patient to raise the arms laterally to a horizontal position. Inability to do so indicates deltoid paralysis. (Circumflex nerves; fourth, fifth, and sixth cervical segments.)

Pectoral Muscles.—Stretch out the arms straight in front, and then approximate the hands against resistance by the examiner, meanwhile watching both heads of the pectoral muscle. (Anterior thoracic nerve; fifth, sixth, and seventh cervical segments.)

Latissimus Dorsi.—Raise the arms laterally to a level; then, while keeping them fully extended, bring the arms downward and backward, as if to make the hands meet behind the sacrum. The examiner standing behind the patient resists the movement. (Subscapular nerves and branches of dorsal and lumbar nerves; sixth and seventh cervical segments.)

Serratus Magnus.—Desire the patient to push with his hands against those of the examiner. If the serratus has lost its power the scapula will project, and the digitations of the muscle, which ordinarily should be visible, will not be seen. (Posterior thoracic nerve; fifth and sixth cervical segments.)

Trapezius.—Ask the patient to raise the shoulders as close to his ears as possible against the resistance of the examiner's hands. This will demonstrate the strength of the upper part of the trapezius. The middle and lower portions are tested by desiring him to bring the scapulæ as close together as possible. (Spinal accessory nerve; medulla and second and third cervical segments.)

It is hardly possible to detect paralysis of the levator anguli scapulæ and rhomboids unless the trapezius is also involved.

Biceps.—Let the patient flex his extended arm, his elbow resting on the observer's left hand, while the latter's right hand, grasping the wrist of the patient, offers the necessary resistance. Also supinate the hand against resistance. (Musculo-cutaneous nerve; fourth, fifth, and sixth cervical segments.)

Triceps.—The triceps may be tested as is the biceps, excepting that the previously flexed arm is to be extended against resistance. (Musculo-spiral nerve; sixth, seventh, and eighth cervical segments.)

Supinator Longus.—Test as for the biceps, except that the hand should be midway between supination and pronation. If the muscle is paralyzed, it will fail to become conspicuous on the radial side of the upper part of the forearm. (Musculo-spiral nerve; fourth and fifth cervical segments.)

Flexors of the Wrist.—Grasping the patient's hand, the palm being upward, desire him to bend the hand up toward his forearm against resistance. (Median and ulnar nerves; eighth cervical segment.)

Extensors of the Wrist.—The patient's hand being held palm downward, he is required to bend it backward against resistance. Moderate weakness of the extensors of the wrist may be manifested by asking him to squeeze the examiner's hand, in which case the wrist will become involuntarily flexed, the weakened extension being unable to counteract the flexors. Marked or complete paralysis of the extensors causes "wrist-drop." (Musculo-spiral nerve; seventh cervical segment.)

Flexors of the Fingers.—Because of the usual difference in the strength of the two hands, the examiner should cross his forearms and place his right hand in the right hand of the patient, and *vice versa*. Then let the patient squeeze his hands. If the observer keeps his own fingers extended and bunched loosely together, he will be able to withstand a very hearty grasp without discomfort. (Median and ulnar nerve; eighth cervical segment.)

Adductor Pollicis.—Ask the patient to pinch, with his thumb and forefinger, one of the examiner's fingers. (Ulnar nerve; eighth cervical segment.)

Opponens Pollicis.—Desire the patient to approximate the ends of the little finger and the thumb. (Median nerve; first dorsal segment.)

The *interosseous* and *lumbrical* muscles of the hand flex the proximal phalanges, and extend the middle and terminal phalanges. The dorsal in-

terossei abduct, the palmar adduct, the fingers from and toward a longitudinal line down through the centre of the middle finger. Test by making the patient separate and approximate the fingers, and flex the proximal phalanges, keeping the middle and terminal phalanges extended. Paralysis of these muscles causes the "claw-hand." (Ulnar and median nerves; eighth cervical and first dorsal segments.)

(3) **Trunk muscles.** Paralysis of the diaphragm is indicated by absence of the protrusion of the epigastrium, which is normally seen during inspiration; instead of protruding, the epigastric region and upper abdominal walls often appear to be sucked in during inspiration. (Pneumogastric nerve and phrenic plexus of the sympathetic.)

The *erector* muscles of the spine are examined by having the patient lie face downward, and asking him to raise the head and shoulders without assistance from the hands. Unless paralyzed, the erectors become plainly visible during the attempt. (Dorsal nerves; second to twelfth dorsal segments.) The *abdominal* muscles are tested in a similar manner, except that the patient lies in the dorsal position while making an effort to raise the head. (Six lower dorsal nerves and lumbar plexus; seventh dorsal to fourth lumbar segments.)

(4) **Muscles of the Lower Extremity.** *Flexors of Thigh.*—The patient lying upon his back, ask him to raise the leg up from the bed, the knee being kept straight. This determines the strength mainly of the ileo-psoas, partly of the quadriceps. (Crural nerve of lumbar plexus; third and fourth lumbar segments.)

Extensors of the Thigh.—The leg being kept straight and the patient upon his back, raise the foot and ask him to bring it down upon the leg against resistance. This determines the strength of the gluteus maximus and partly of the hamstring muscles. (Superior and inferior gluteal nerves, first and second sacral segments; and the sciatic nerve, fifth lumbar segment.)

Abductors of the Thigh.—Carry the leg across the middle line, and desire the patient to force it toward the outer side against resistance, thus testing mainly the gluteus medius. (Superior gluteal nerve from the sacral plexus; first and second segments.)

Adductors of the Thigh.—Carry the leg outward and desire the patient to force it back to the middle line against resistance, thus testing the adductors longus, brevis, and magnus. (Obturator, great sciatic and crural nerves; from lumbar plexus.)

In-rotators of the Thigh.—With the patient lying face downward, flex the knee to a right angle, grasp the foot, and oppose resistance while he in-rotates the thigh. This tests mainly the gluteus minimus. (Superior gluteal nerve from the sacral plexus.)

Out-rotators of the Thigh.—Test the power of out-rotation in similar

fashion, thus determining the condition of the pyriformis, gemelli, quadratus femoris, and obturators. (Sacral and lumbar plexus.).

Flexors of Knee.—With the patient lying face downward, or even while he sits in a chair, let him bend the knee while the examiner resists the movement by pressure against the heel. This tests mainly the biceps, semi-membranosus, and semi-tendinosus. (Sciatic nerve; fifth lumbar segment.)

Extensors of Knee.—With the patient in the dorsal position, flex the knee and by pressure upon the sole of the foot resist his endeavor to extend the knee, thus testing principally the quadriceps femoris. (Crural nerve; third lumbar segment.)

Plantar Flexors (Extensors) of the Foot.—With the leg straight, resist by pressure upon the sole the patient's endeavor to bring the tarsus in line with the leg, thus testing the gastrocnemius, soleus, peroneus longus and brevis. (Internal popliteal and peroneal nerves; fifth lumbar and first and second sacral segments.)

Dorsiflectors of the Foot.—With the leg straight, resist the patient's attempt to bend up the foot, thus testing the tibialis anticus and the peroneus tertius. Marked paralysis of these muscles causes "foot drop." (Anterior tibial nerve; fifth lumbar and first sacral segments.)

Muscles of the Foot.—The flexors, extensors, interossei, and lumbricals of the toes are examined in a manner similar to that applied to the hand. (Posterior tibial nerve; first and second sacral segments.)

Paralysis.—Having ascertained by the above tests the presence of paralysis, it becomes essential to determine its **type,** *i. e.*, whether cerebral, due to a lesion of the upper neuron, or spinal, the result of disease of the lower neuron. The motor path consists of two segments: the *upper neuron*, whose axon leaves its cell in the cerebral cortex and joins with others to form the motor fibres which traverse the corona radiata, internal capsule, crus cerebri, pons, and medulla, and then for the most part cross to the opposite side and pass down the crossed pyramidal tracts to terminate in the gray matter of the anterior cornua; and the *lower neuron*, which commences in the cells of the anterior cornua and is prolonged as a part of a peripheral nerve to its termination in the muscles. These two neurons, although in close contiguity, are independent of each other, and the nutritional welfare of each depends entirely upon the integrity of its own cell body. Therefore degeneration due to disease of the upper neuron does not extend beyond its own terminal fibres and the lower neuron remains

intact, and *vice versa*. So long as the disease is limited to the upper neuron, the muscles undergo no wasting (except a slight atrophy from disuse) and the electrical reactions are not definitely altered. If, however, the lower neuron is affected, the power of movement will be absolutely lost, the muscles will become flaccid and atrophied, the reflexes will be lost, and the electrical reactions markedly changed.

Clinically we may distinguish these two types of paralysis by the following signs:

(1) The **Cerebral Type,** of which hemiplegia is characteristic, is marked by *spasticity* of the muscles, the limb resisting attempts at passive movement or undergoing a spasmodic contraction when the latter is attempted. *Contractures* may develop; the *reflexes are exaggerated*, owing to absence of the inhibition normally exercised by the upper neuron; the *electrical reactions are normal* and there is *no atrophy*.

(2) The **Spinal Type,** of which paraplegia is characteristic, is indicated by a *flaccid* state, the member being limp and the muscles appearing lax and offering no resistance to passive movement. The *reflexes are diminished or lost;* there is partial or complete loss of response to the faradic current, with partial or entire *reaction of degeneration* to the galvanic current; and there is *marked atrophy*.

The cerebral type of paralysis is due to a lesion of the motor cortex, of which the pyramidal tract is an extension, and is usually unilateral (hemiplegia), rarely bilateral (diplegia). It may be due to disease of the crossed pyramidal tract in the cord (lateral sclerosis). The spinal type is the result of lesions affecting the cell-bodies in the medulla or pons (*e. g.*, bulbar paralysis), of the motor cells in the anterior cornua of the cord (*e. g.*, poliomyelitis), or of the peripheral nerves (*e. g.*, neuritis). A mixed type may be the result of a simultaneous involvement of the terminals of the upper neuron and the cell-bodies of the lower neuron (amyotrophic lateral sclerosis, transverse myelitis). Functional paralysis (hysteria) is usually flaccid and presents neither atrophy nor the reaction of de-

generation. As a rule, the history and the presence of hysterical stigmata will serve to distinguish this latter condition.

Localization and Nature of the Lesion.—Complete hemiplegia, unless functional, is always due to a cerebral lesion. Hemiplegia with motor aphasia, in a right-handed person, is caused by a lesion of the third left frontal convolution or by a lesion in the capsule interrupting the fibres therefrom. Hemiplegia with lower facial palsy indicates a lesion in the posterior half of the internal capsule. Hemiplegia with an opposite (crossed, alternating) ocular paralysis is due to a lesion in the crus. Hemiplegia with opposite facial palsy indicates a lesion of the pons. Hemiplegia with difficulty in articulation and deglutition (not aphasia) is due to a lesion of the medulla. Diplegia is the result of a bilateral or symmetrical cerebral lesion, and occurs principally in children.

As a rule, hemiplegia is the result of cerebral hemorrhage, thrombosis, or embolism; it is sudden in onset, but is often preceded by arterio-sclerosis, and it is followed by softening. Uremia occasions a transient hemiplegia, hysteria a functional variety; and a gradual, progressive hemiplegia may be due to a brain tumor or an island of sclerosis.

Spastic paraplegia, if the sphincters are involved or the reflexes exaggerated, is certainly due to a lesion of the cord. It may be due to spinal meningitis, Pott's disease, hereditary ataxic paraplegia, lateral sclerosis, disseminated sclerosis, chronic myelitis, transverse myelitis, syphilis, or the spastic cerebral paralysis of children. Flaccid paraplegia may be due to spinal disease (Friedreich's ataxia, acute poliomyelitis, intra-spinal hemorrhage, tumor, or tuberculosis) or to neuritis.

Monoplegia, if cerebral, is almost invariably cortical or slightly sub-cortical; it may be due to hemorrhage, embolism, tumor, etc., and the paralysis is of cerebral type, *i. e.*, spastic, non-atrophic. If spinal, a monoplegia is usually due to poliomyelitis or unilateral myelitis. More often the cause is peripheral, *i. e.*, neuritis, and is distinguished by its limitation to the muscles supplied by a certain nerve. When neuritis is

widespread, however, it may be difficult or impossible, because it is a disease of the lower neuron, to exclude a lesion of the anterior horn of the cord.

2. **The state of the muscular nutrition.** Pinch the muscles and note whether they are firm or wasted and flabby. This is a rough test, but if only one limb is affected, comparison with the other renders it effective.

A more delicate test of the muscular nutrition is afforded by investigation as to their **electric irritability.** For this purpose it it necessary to have a galvanic and a faradic battery, together with a rheostat, a milliamperemeter, and a switch for changing the direction of the galvanic current. Begin with the faradic current. The examiner should first test the strength of the latter upon his own wrist, in order to be sure that it is very weak at the start. Place a large (indifferent) electrode between the shoulders or on the chest or abdomen, and apply a small (normal) electrode upon the nerve or motor points of the muscle to be examined. Note the minimum intensity of current (indicated by the distance to which the inner and outer coils overlap) which produces contraction at each point, and compare it with the effects of a similar current upon the corresponding point on the other side of the body.

Then use the galvanic current. Place the electrodes in exactly the same positions as before, and arrange the switch so that the small electrode becomes the negative pole or cathode. Turn on the current and let it run for a moment; and open and close the circuit in this way several times, while slightly increasing or decreasing its strength, until the minimum force which will cause a muscular contraction at the instant of closing the circuit has been ascertained. This represents the cathodal closing contraction (CaCC). Note the strength of current indicated on the milliamperemeter.

Reverse the direction of the current, thus making the small electrode the positive pole or anode. Repeat the same tests, thus ascertaining the anodal closing contracture (AnCC). By using the same strength of current in each case, determine whether CaCC is stronger or weaker than AnCC. The char-

acter of the muscular contraction is also of great significance, *e. g.*, quick and sharp, slow or sluggish, or continuous and tetanic.

Exceptionally it is necessary to make similar tests with the opening of the cathodal circuit (CaOC) and of the anodal (AnOC).

The Reaction to Electrical Stimulation.—In health a sharp contraction follows closure of the faradic current and continues as long as the passage of the latter. When the galvanic current is applied, contractions appear only at the opening and closing of the circuit. Normally, CaCC is the first to appear, and a decidedly strong current is necessary to induce AnCC. Still further increase of current is necessary to elicit AnOC and CaOC. Moreover, in health the muscular contractions are sharp and abrupt.

In disease these reactions to electrical stimulation may undergo either quantitative or qualitative alterations. *Quantitative alterations* are present when a given current produces greater or less contraction than it would were the nerves and muscles normal. *Qualitative alterations* are indicated by a change in the character of the contraction, the latter becoming sluggish, or the cathodal closing contraction being more difficult to elicit than the anodal contractions. In their typical form these alterations constitute the **reaction of degeneration.** This is indicated as follows:

1. With the faradic current—no response.

2. With the galvanic current—sluggish response (extremely significant), and a response to the positive pole which is as good or even better than that to the negative pole.

Significance of Altered Electrical Responses.—These changes are due to separation of the nerve and muscle from their nutritive centre, the cell body. In other words, a lesion of the lower neuron is present. If the reaction of degeneration is obtained, the examiner can exclude cerebral disease, functional paralysis, and muscular dystrophy; and he can positively assert that some disease of the lower neuron (anterior cornua and roots or peripheral nerves) is present.

3. **Muscular co-ordination.** The harmonious co-operative action of groups of muscles is necessary to perfect movement or station, and through the disturbance of this harmony certain acts become difficult. Inco-ordination (*ataxia*) may be due to absence of vision, to disturbance of cutaneous sensibility, or to loss of the muscular, articular, and tendinous senses whereby the sensorium is cognizant of the position of the various limbs and parts of the body.

Tests.—Upper limbs. Bandage the eyes and then ask the patient to touch the tip of his nose first with one forefinger, then with the other; or require him to bring the tips of his two forefingers together. If he is able to do these things, no inco-ordination is present.

Lower limbs. Request the patient to walk along a straight line, such as a crack in the floor; or if he is lying in bed, ask him to touch the dorsum of one foot with the great toe of the other. Inability to do this constitutes *motor ataxia.*

Require the patient to stand with his heels together and his eyes closed, and note whether he sways markedly. If he does, *static ataxia* (Romberg's symptom) is present.

Cerebellar ataxia, due to disturbance of equilibration by cerebellar disease, is characterized by a reeling, drunken gait. If the patient is in bed, he can successfully co-ordinate his lower limbs, imitating, with his foot, movements (circles in the air, etc.) made by the examiner's hand, touching stated points with his great toe, etc. In addition, the arms do not become ataxic.

4. **Abnormal muscular movements.** Examination of the motor functions may reveal an abnormal degree of muscular contraction, *i. e.*, *spasm.* If this spasm is painful, it is called a *cramp.* If it consists of a more or less continuous contraction, the spasm is *tonic.* A paralyzed muscle which is constantly in a state of tonic spasm is said to be *spastic*, and this muscular contraction if long-continued leads to a permanent *contracture.*

On the other hand, there may be alternate contraction and relaxation of the muscle (*clonic spasm*), and if this action

involves a majority of the muscles, a "fit" or *convulsion* is said to be present. Both clonic and tonic spasms are seen in the convulsions of children and those of epilepsy, uremia and hysteria. Pure tonic spasms are present in tetanus and tetany.

Tremor is the simplest form of clonic spasm. It consists of a trembling movement, especially of the extremities, and sometimes affects the eye ball (*nystagmus*). According to the amplitude of the movements a tremor is said to be *fine* or *coarse*, and it may be slow or rapid. The movements may be present while the muscle is at rest (*passive tremor*), or it may begin or increase when voluntary movement is attempted (*intention tremor*). The tremor may involve the entire body or a single limb or a group of muscles. Exceptionally, only a few muscle fibres are affected (*fibrillation*), and this fibrillary spasm may be seen as a few irregular twitchings under the skin (progressive muscular atrophy, neurasthenia).

Tests.—Let the patient stretch out his arms with the fingers extended and as wide apart as possible. If the tremor cannot then be seen, touch the patient's fingers with the hand and a thrill may be felt. Tremor of the tongue or face is best exhibited when the patient closes his eyes and protrudes his tongue vigorously for a few moments. Intention tremor is brought out by having the patient attempt some delicate coordinate movement, such as carrying a cup to his lips.

Tremor may be merely a sign of excitement in nervous people. A persistent tremor is characteristic of certain diseases, notably exophthalmic goitre, disseminated sclerosis, and paralysis agitans; and it occurs with less regularity in senility and in alcoholic and other intoxications.

Athetosis.—Athetoid movements consist of continuous, unceasing twisting motions of the fingers and hands, sometimes the toes and feet. They are occasionally seen in hemiplegics.

Convulsions may be *general*, involving the entire body, or *local*, confined to a limited area, such as the face or one extremity. A local convulsion which gradually extends to the adjacent muscles, with slight or tardy loss of consciousness, is

called *Jacksonian*, and usually indicates irritation of a particular point in the cerebral cortex, the exact location of which may be judged by the initial spasm (*signal symptom*).

Choreic movements consist of abrupt, jerking motions, involuntary and objectless, of certain muscle-groups. They are seen not only in chorea but in a number of other affections (*e. g.*, Huntingdon's chorea, habit chorea), and are sometimes induced by the intense toxemia of an acute infectious disease.

IV. SENSORY FUNCTIONS.

The investigation of the sensory functions of a patient should include, in addition to sight, hearing, smell, and taste (see *Cranial Nerve Functions*), tests for common sensibility (touch and pressure), and the pain, temperature, and muscular senses. For this examination the patient should be blindfolded and instructed to maintain silence, signifying by monosyllables his responses to the tests applied. Note not only the correctness but the promptness of his replies.

1. **Common sensibility.** (*a*) **Tactile sense.** For delicate testing the esthesiometer, which is virtually a pair of compasses with blunt points, may be used. For practical purposes, however, it is enough to draw the tip of the finger, the end of a pencil, or a bit of cotton lightly across the patient's skin, asking him to say "now" when he feels it. Compare corresponding points on the opposite sides of the body, and occasionally omit the touch, in order to prevent unthinking responses from the patient. Ask him also to point out the exact spot touched; if he errs by more than two inches, slight anesthesia is present.

Tactile sensibility may be abolished (*anesthesia*) as the result of interruption to the sensory paths at any point between the periphery and the brain. This may be in the cerebrum (especially lesions of the sensory portion of the internal capsule), the posterior nerve roots (tabes dorsalis), or the peripheral nerves (neuritis). Anesthesia, often of

peculiar distribution, constitutes one of the stigmata of hysteria. Increased tactile sensibility (*hyperesthesia*), as a result of which a mere touch may be painful, is often present over hysterogenic zones. It also occurs in connection with meningitis, neuritis, and certain other diseases.

(*b*) **Pressure sense.** The sense of pressure may be tested by supporting the part to be examined upon a bed or other support, and placing upon it objects similar in size, but differing in weight (*e. g.*, coins), and asking the patient to say which is the heavier. Loss of the pressure sense, tactile sensation remaining, may be observed in tabes dorsalis.

2. **Pain sense.** The point of a pen or a sharpened pencil may be employed to test the acuteness of the pain sense, the examiner touching the symmetrical parts. It may be absent (*analgesia*) in syringomyelia, Morvan's disease, and hysteria; it is usually exaggerated (*hyperalgesia*) in association with hyperesthesia.

3. **Temperature sense.** Fill two test tubes, one with hot and the other with cold water. Touch the skin with each in turn, asking the patient whether it feels hot or cold, and note whether he answers correctly. The temperature sense is lost in syringomyelia, and to a lesser degree in tabes dorsalis and diseases of the medulla.

4. **The muscular sense,** with which are included the articular and tendinous senses, may be tested in two ways. (*a*) Weights, such as were employed in testing the pressure sense, are placed over the part to be tested, which in this case should be without support. The patient is requested to say which weight is the heavier. (*b*) The examiner moves one of the patient's extremities, flexing or extending it, and requests the patient to move the opposite limb in a similar fashion. Or, one limb may be placed in a certain posture and the patient asked to describe it. Impairment or loss of the muscular sense distinguishes ordinary from cerebellar ataxia; it is associated with some varieties of cerebral diseases and with tabes dorsalis.

Abnormal sensations (*paresthesiæ*) may be present but require

no tests for their discovery. The commonest of them are numbness, "pins and needles," constriction, itching (*pruritus*), and a sensation as of insects crawling over the skin (*formication*).

Delayed sensation, the response of the patient being delayed far beyond the normal one-tenth of a second, may be associated with tabes dorsalis and various peripheral palsies.

V. REFLEX FUNCTIONS.

For the production of a reflex it is necessary for a stimulus to travel from the periphery along an afferent nerve to the motor cells in the cord or medulla, where it is transferred into an impulse which traverses an efferent (motor) nerve to certain muscles and induces their involuntary contraction. Three classes of reflexes are recognized, viz.,

1. **Superficial (cutaneous) reflexes.** The following depend upon centres in the medulla:

The Conjunctival Reflex.—Touch the conjunctiva and the eyelids close abruptly. The fifth is the afferent and the seventh the efferent nerve.

Pupillary-Skin Reflex.—Stroke the skin of the chin or neck, and the pupils dilate.

Palatal Reflex.—Touch the mucous membrane of the palate and the latter draws up. The ninth is the afferent and tenth and eleventh the efferent nerves.

The more important reflexes dependent upon the spinal cord are as follows:

Plantar.—Stroking the sole of the foot is followed by movements of the toes, foot, or leg. (Lower part of lumbar enlargement.)

Gluteal.—Stroking the skin of the buttock is followed by contraction of the gluteal muscles. (Fourth and fifth lumbar segments.)

Cremasteric.—Stroking the skin at the upper and inner part of the thigh is followed by retraction of the testicle. (First and second lumbar segments.)

Abdominal.—Stroking the abdominal wall downward from the costal margin in the nipple line causes contraction of the abdominal muscles. (Eighth to twelfth dorsal segments.)

Epigastric.—Stroking the side of the chest downward from the nipple is

followed by a retraction of the epigastrium on the same side. (Fourth to seventh dorsal segments.)

Scapular.—Stroking the skin in the inter-scapular region causes contraction of the scapular muscles. (Fifth cervical to first dorsal segments.)

The significance of a superficial reflex is practically limited to a demonstration of the integrity of the reflex arc (sensory nerve, spinal segment, motor nerve) upon which it depends.

2. **Deep (tendon) reflexes.**—When a muscle is put upon the stretch and its tendon sharply struck, it contracts abruptly. The more important of the reflexes thus obtained are the following:

The Jaw Jerk.—Place a ruler on the lower incisor teeth, or the finger upon the chin, while the mouth is partially opened, and upon the ruler or finger deliver a sharp percussion stroke. The masseter and temporal muscles contract sharply, closing the jaw. (Motor nucleus of fifth nerve.)

The Elbow or Triceps Jerk.—With the elbow flexed, the arm resting upon the examiner's hand or upon the back of a chair, and the forearm hanging limply downward at a right angle, tap just above the point of the elbow. The forearm performs a quick movement of extension.

The Wrist Jerk.—With the hand hanging down as in wrist-drop, strike the extensor tendons near the wrist joint. The hand is suddenly extended,

The Knee Jerk.—The knees may be crossed, the patient may sit on a chair or table high enough to keep his feet from the floor, or the examiner may support the leg by placing his hand or arm under the crook of the knee. The leg must hang loosely. Then strike a sharp blow on the tendon just above the patella. This should be followed by a sharp jerk of the leg and foot. If the latter does not occur, require the patient to hook his hands together and pull with all his strength ("reinforcement"); under these circumstances the knee jerk can, in health, almost always be elicited.

The Ankle Jerk.—Extend the patient's leg and grasp the foot, bending the latter upward so as to put the tendo achilles

on the stretch. Strike the latter sharply, and notice the resulting contraction of the calf muscles.

Ankle Clonus.—With the leg partially extended and the knee slightly bent, hold the heel on the left hand and with the right abruptly push up the front part of the foot toward the leg and press steadily. Under certain abnormal circumstances a rhythmic, clonic movement ensues, and is continued for some time.

Significance of the Tendon Reflexes.—The deep reflexes of the upper extremity may be absent in health. The jaw jerk is always abnormal; the presence of it and the elbow and wrist jerk is strongly suggestive of central disease. The knee jerk is almost always present in health, but if any portion of its arc is interrupted, it is abolished. *Loss of the knee jerks* may, therefore, be due to disease of the peripheral nerves (neuritis), disease of the posterior roots or columns (tabes dorsalis, Friedreich's ataxia), disease of the anterior cornua (poliomyelitis, Landry's paralysis), or to myelitis affecting the second and third lumbar segments. *Exaggeration of the knee jerk* is due to lessened inhibition by the upper motor neuron, which may be due to paralysis of cerebral origin involving the pyramidal tracts, hereditary cerebellar ataxia, paralytic dementia, lateral sclerosis, amyotrophic lateral sclerosis, and myelitis. It may also be a symptom of hysteria, neurasthenia, arthritis deformans, tetanus, and strychnine-poisoning.

The ankle jerk corresponds in significance to the knee jerk. Ankle clonus is present under the conditions which cause exaggeration of the knee jerk; it is particularly marked in disseminated and amyotrophic lateral sclerosis.

3. **Organic reflexes.** Respiration, deglutition, micturition, and defecation depend upon complex muscular movements of reflex origin. Difficulty or pain in urination or defecation, and incontinence of either urine or feces may, therefore, possess great diagnostic significance. The centres controlling the rectum and bladder are situated in the fourth and fifth sacral segments.

CEREBRAL LOCALIZATION.

The following (Strümpell) affords a convenient summary of the main facts bearing on the localization of cerebral disease:

(1) The most frequent cause of ordinary hemiplegia is a lesion of the *pyramidal* tract in the posterior limb of the internal capsule. If the hemiplegia be persistent, then this tract is actually destroyed; if temporary, the tract has been functionally deranged for a time by focal disease in the neighboring parts of the brain.

(2) Monoplegic cerebral paralysis is usually due to affections of the *cortex* of the brain, that is, the central convolutions and the paracentral lobule. Monoplegia of the face and tongue is the result of lesions in the lower extremity of the anterior central convolution. Monoplegia of the arm is referable principally to some lesion of the middle third of the anterior central convolution. Monoplegia of the lower extremity implies some affection of the paracentral lobule.

(3) Hemiplegia or monoplegia, if associated with epileptiform convulsions affecting either one-half or one particular portion of the body, are almost always caused by cortical lesions. These same symptoms of motor irritation with accompanying paralysis are likewise ascribable to some irritation of the above-mentioned regions of the cortex.

(4) Hemiplegia with crossed paralysis of the oculomotor nerve indicates a lesion of the crura cerebri.

(5) Hemiplegia with crossed facial paralysis implies, with an approach to certainty, that the lesion is situated in the pons.

(6) Post-hemiplegic chorea seems to occur especially where there is focal disease in the neighborhood of the optic thalamus or of the posterior part of the internal capsule.

(7) Hemianesthesia seems to result principally from lesions of the most posterior portion of the internal capsule.

(8) Hemiopia may be due to a lesion of the occipital lobe. Probably, also, a lesion of the posterior extremity of the in-

ternal capsule may cause it, in which case it is usually associated with hemianesthesia. Finally, it may be produced by lesions of the posterior tubercle of the optic thalamus, or one of the anterior corpora quadrigemina, or of one of the optic tracts.

(9) Genuine motor aphasia indicates disease of the 3d left frontal convolution.

(10) Word-deafness seems to be due to disease of the 1st left temporal convolution.

(11) Difficulty in articulation implies disease of the medulla, as does also dysphagia.

(12) Staggering gait and vertigo are the most constant symptoms of cerebellar disease. Forced positions and forced movements are seen chiefly in connection with lesions of the crura cerebelli ad pontem.

(13) Staggering gait and ocular paralysis are indicative of lesions of the corpora quadrigemina.

DISEASES OF THE BRAIN AND ITS MEMBRANES.

PACHYMENINGITIS.

Inflammation of the dura mater may affect its outer surface (*pachymeningitis externa*) or its inner surface (*pachymeningitis interna*).

Pachymeningitis externa is due to infection spreading from adjacent structures (fracture of the cranium with sepsis, osteitis, necrosis, neoplasms, and suppurative middle ear disease). The dura becomes thickened, with adhesion to the inner surface of the cranium, and sinus-thrombosis or septicemia may ensue. Treatment should be directed to the primary condition.

Pachymeningitis interna (pachymeningitis hemorrhagica, hematoma of the dura mater) is a chronic inflammation of the inner surface of the dura attended by hemorrhagic extravasations between its layers, the effused blood becoming organized

into laminated new membranes. It is due to vascular degeneration, the result of alcoholism, infectious diseases, and cachectic conditions; and it is a constant association of paralytic dementia. The symptoms are vague until pressure produces cerebral manifestations, such as pain in the head, intellectual failure, Jacksonian convulsions, apoplectiform seizures, transitory hemiplegias, and rarely vomiting. The duration of the disease is variable, but it tends to death. Diagnosis is extremely difficult, but the occurrence of the symptoms enumerated in drunkards or paretics may suggest the disease. Treatment should as far as possible be directed to the casual condition.

LEPTOMENINGITIS.

Etiology.—Inflammation of the pia mater (acute cerebral meningitis, purulent meningitis) may result from lesions of neighboring structures (cranial fractures, and septic infections of the scalp, face, middle ear, orbit, nares, etc.), or from microbic infection through the vascular supply (pneumonia, rheumatic fever, and other infections; coryza). The epidemic cerebro-spinal variety, due to the diplococcus intracellularis, has been described elsewhere.

Pathology.—The infective agents (pneumococci, streptococci, typhoid bacilli, staphylococci, etc.) may set up a localized inflammation by extension from an adjacent focus; but when the infection occurs through the blood, the meningitis is generalized, though most intense over the vertex. The arteries are engorged, and the membrane is roughened and more or less covered with a purulent exudate; the ventricles are dilated with the increased fluid, and the cerebral cortex is edematous, infiltrated with pus, and adherent to the pia.

Symptoms.—The disease may be preceded by several days of malaise, feverishness and slight headache. A rigor may accompany the onset, and four cardinal symptoms develop almost immediately, viz.,

1. *Fever.* While constant, this is rarely high (100–103°).

2. *Headache.* This is violent and subject to paroxysmal exacerbations. Even when comatose the patient, by holding the head in the hands, by groans, and by facial expression, indicates its continued presence.

3. *Delirium.* This is usually of a low type, but occasionally it is wild and maniacal.

4. *Vomiting.* This is projectile in character and unaccompanied by nausea.

Associated with these symptoms there may be muscular rigidity, which in the neck is associated with retraction, and convulsions, particularly in children. Sooner or later the cranial nerves become involved, and as a result there may be photophobia, neuro-retinitis, strabismus, myosis, mydriasis, or immobile pupils, and deafness. Cutaneous hyperesthesia is present over the trunk and limbs, and if the finger nail be drawn across the skin of the abdomen, it is followed slowly by a red streak that persists for some minutes (*tache cerebrale*). The pulse may be full and active early in the attack, but later it is sluggish and slow (60–40), thus showing disassociation with the temperature. The bowels are constipated and the abdomen "scow-shaped."

Course.—The disease may end fatally within twenty-four hours, or it may extend over several weeks. In the average case a week of active symptoms merges into a state of depression, with deepening coma and finally death from heart failure or bulbar involvement.

Chronic Leptomeningitis may be a sequence of the acute variety, or it may be due to alcoholism, syphilis, or sunstroke. Clinically, except in the syphilitic form, its manifestations are so obscure as to render diagnosis impossible.

Diagnosis.—In the presence of causal conditions, the appearance of the four cardinal symptoms mentioned will justify a tentative diagnosis of meningitis. *Uremia* should be excluded by careful urinalysis.

Prognosis.—The mortality varies from 30 to 60 per cent., and even should recovery take place, permanent brain injury

is apt to result. In no case can the outcome be foretold by the symptoms; the mildest attack may end fatally, the worst recover.

Treatment.—Remove, if possible, the infective focus; render aseptic any cavity, cranial, optic, nasal, pharyngeal, intestinal, or pelvic, the contents of which justify suspicion. With this in view it is wise to empty the bowels by enemata or cathartics. Of medicines, *aconite* may be indicated by the early febrile symptoms, *arnica* by a history of traumatism, and *belladonna* by violent headache and delirium. The development of effusion is accompanied by symptoms which suggest the administration of *bryonia*. The convulsive phenomena may be met by the use of *cuprum* or *cicuta*. Among remedies less frequently useful are *apis*, *gelsemium*, *glonoin*, *helleborus*, *mercurius*, *œnanthe crocata*, *sulphur*, and *zinc*.

HYDROCEPHALUS.

Etiology.—Increase of the quantity of cerebro-spinal fluid may occur within the ventricles (*internal hydrocephalus*) or in the sub-dural spaces (*external hydrocephalus*). It may be acute or chronic, congenital or acquired; but in a vast majority of cases it appears at birth or within the first six months of life. The causes are obscure, although parental syphilis and alcoholism are important factors, and meningitis, traumatic or otherwise, may induce it.

Pathology.—In the majority of cases occurring before the cranial bones are united the head becomes greatly enlarged, the bones are thin and without diploë, and the sutures are open. The lateral ventricles are often enormously distended, and in half the cases the ventricular aqueducts are obliterated, the third and fourth ventricles thereby escaping. The contained fluid is colorless, of low specific gravity, and resembles other serous effusions. The convolutions and often the basal ganglia are compressed and flattened. Should the disease develop after cranial solidification has taken place, the head does not enlarge and the amount of fluid is less, but the pressure upon the cerebrum is consequently greater.

Symptoms.—Hydrocephalus may render birth difficult and even necessitate the use of the perforator. As a rule, however, it is not noticed until the rapid enlargement of the head during the first year of life attracts attention. The outline of the skull is globular, and it appears to overhang the face, which is not similarly enlarged. The internal pressure is manifested in bulging fontanelles and open sutures, and the superficial veins of the scalp are distended.

The child is inactive, often peevish and restless, and frequently repeats a distressed cry. It raises its head with difficulty or not at all. If death does not soon occur from exhaustion, convulsions, or coma, retarded growth usually manifests itself in a dwarfish body and a mental development which is defective in varying degrees. Optic atrophy and blindness, nystagmus, strabismus, and spasticity of the lower limbs are frequently associated with the other symptoms.

Diagnosis.—The enlarged globular head usually affords a diagnosis. In the only similar condition, *rachitis*, the head is not round, but square, with a bulging forehead, and rickety conditions may be detected in the long bones and at the costal extremities. In the absence of cranial enlargement the disease can be discovered only post mortem.

Hydrocephaloid (hydrencephaloid, spurious hydrocephalus), is a term applied by Marshall Hall to a condition supervening upon infantile diarrheas in which the patient becomes semicomatose, with fingers clutched, head retracted, and respiration irregular. The condition has been attributed to cerebral anemia, followed by venous hyperemia and consequent effusion. It is more probably the result of aute-intoxication. It bears no relation whatever to true hydrocephalus.

Prognosis.—In a vast majority of cases death occurs within a year. A few patients live for several years, but their life is much shortened and their mentality is very poor. In rare cases the disease becomes stationary, and a fair degree of mental and physical development and a long life become possible.

Treatment.—This is very unsatisfactory. When the process is active, cold may be applied to the head. Some success has attended exposure of the child's head to the sun, beginning with twenty minutes each day and increasing to thirty or forty. Strapping the head with adhesive plaster has been tried; but it causes intolerable pain and only serves to increase the intracranial pressure. Repeated tapping through the fontanelles or by lumbar puncture has in a few cases been attended with apparent success; only a small amount of fluid should be withdrawn at one time, and strict antisepsis is necessary. Attempts at permanent drainage have almost invariably been followed by death. A method recently devised consists of the introduction of drainage tubes through a drill opening above and back of the ear; union is then secured between the covering scalp-flaps and in the cavity thus formed the fluid is allowed to accumulate and reabsorb.

Medicines should be prescribed according to the underlying constitutional conditions rather than mere symptoms; the more important are the *calcareas*, *arsenic*, *baryta carb.*, *kali iod.*, *lycopodium*, *mercurius*, and *sulphur*.

CEREBRAL ANEMIA.

Etiology.—Cerebral anemia is invariably secondary. It is often associated with general anemia or cachexia; it may be due to mechanical causes, such as an insufficient heart or occlusion of a carotid by ligation or atheroma; it results from emotional excitement, such as pain or shock; and it can be induced by various poisons, such as tobacco, ergot, chloroform, and the bromides.

Symptoms.—Cerebral anemia may be acute, as in an ordinary fainting fit following great loss of blood. Under such circumstances the patient becomes giddy, hears noises in the ears, everything grows dark before his eyes, and he loses consciousness more or less completely.

In the chronic cerebral anemia attendant upon general anemia, etc., the patient suffers with dull headaches and a

sense of heaviness or constriction about the head. Vertigo, tinnitus, and muscæ volitantes may be noted, especially when he rises suddenly. The mental condition is altered; the patient is inclined to be irritable and peevish, and he is physically inactive.

Diagnosis and **treatment** should be directed to the underlying cause.

CEREBRAL HYPEREMIA.

Etiology.—Acute congestion of the brain occurs normally under the influence of great excitement or unusual mental or physical exertion. It may be caused by certain drugs (nitrites, alcohol), certain infectious diseases (typhoid, typhus, etc.), or by sunstroke. Menstrual suppression, or the menopause, may induce cerebral hyperemia; and overactivity of a hypertrophied heart, or the vascular "storms" of the gouty, may underlie its occurrence.

A chronic cerebral congestion, passive in character, may be the result of heart lesions or other conditions impeding the venous return, or it may be associated with chronic disease of the brain itself.

Symptoms.—Acute cerebral congestion, if marked, causes a throbbing headache, blurred vision, photophobia, vertigo, tinnitus, and frightful dreams during sleep. Delirium or convulsions may supervene, or even an apoplectiform attack may be induced. Chronic hyperemia is accompanied by a sense of fulness, a dull frontal headache, and various mental symptoms.

Diagnosis.—The sense of fulness in the head associated with flushed face, throbbing arteries, and aggravation when the head is lowered, is usually sufficient to distinguish cerebral congestion. It is necessary, however, to discover its cause, and to that **treatment** must be directed.

INTRACRANIAL HEMORRHAGE.

(Cerebral Apoplexy.)

Etiology.—Hemorrhage into the substance of the brain is the direct result of the coincidence of two factors, viz.,

1. *High arterial tension.* A strong, forcible heart driving the blood into inelastic arteries produces high tension within the cerebral arteries. The hemorrhage may, therefore, be directly induced by emotional excitement, over-exertion, coughing, sneezing, vomiting, or chilling of the surface of the body, all of which tend to force more blood into the cerebral circulation.

2. *Lessened resistance of the arterial walls.* Arterial disease, inflammatory or degenerative, may be associated with gout, syphilis, alcoholism, plumbism, or chronic nephritis. This arterial condition is usually present in the aged, and in childhood the arteries are naturally weak; consequently it is in early life and advanced age that intracranial hemorrhage is common. In some cases there appears to be a hereditary tendency to cerebral hemorrhage.

Pathology.—All parts of the brain are subject to arterial disease and in consequence thereof to hemorrhage; but by far the most frequent site of the latter is the area about the lenticulostriate branches which arise from the trunk of the middle cerebral artery and supply the basal ganglia and the internal and external capsule. Next in order of frequency are the centrum ovale, cortex, cerebellum, pons, medulla, crura, and corpus callosum. Usually by the rupture of a miliary aneurism in the vessel wall, blood is extravasated, and the latter separates or breaks through the tissues adjacent to the site of the hemorrhage. There later it undergoes clotting, the serum is absorbed, and the remaining ocherous mass, consisting of blood-crystals, pigment and fatty detritus, may become encysted and even cicatrized. The motor tracts intercepted by the lesion undergo a degeneration which extends down the entire length of

the neuron, and involves the lateral tracts of the cord, especially on the side opposite the lesion.

Symptoms.—Rarely momentary confusion or vertigo precedes the attack, but as a rule the onset is abrupt—a "stroke." Consciousness is almost instantly abolished, and the patient, if upright, falls. At first the face is pale, the pupils contracted, and muscular twitchings or even general convulsions may occur. In the "apoplectic" state which ensues the patient lies inert, unconscious, and his breathing may be slow, stertorous, or of the Cheyne-Stokes type. His face becomes congested, all forms of sensibility are abolished, and the sphincters are relaxed. It is often difficult to detect the paralysis at this time, but already the patient tends to direct his face and eyes to the side of the brain that contains the hemorrhage (*conjugate deviation*), the arm or leg of the paralyzed side, if lifted, falls limply, and whatever slight movements the patient makes are restricted to the sound side.

This coma may deepen into death, or it may pass off. In the latter case, a period of torpor ensues, from which the patient can temporarily be aroused by painful stimulation, such as pinching. Then he begins to hear loud voice sounds, and finally by facial expression and attempts at speech he indicates his partial return to consciousness. For some hours he is still inclined, if undisturbed, to relapse into stertorous sleep; but finally consciousness becomes completely restored.

The *Hemiplegic state* which follows intracranial hemorrhage may also, it must be remembered, result from traumatic lesions of brain and cord, from meningeal lesions (hemorrhage, inflammation, syphilis and tuberculosis), from cerebral lesions (softening, tumors, abscesses, and sclerosis), and from cerebro-spinal lesions (locomotor ataxia, disseminated sclerosis, and paretic dementia). In cases of hemiplegia due to intracranial hemorrhage, if death does not occur within three weeks after the onset, the hemiplegia begins to improve and continues to do so steadily for several months. The exact degree of paralysis that will persist cannot, however, be foretold; in some cases it remains extreme, in

others it becomes very slight. At first the reflexes are abolished and the muscles limp, but gradually, as the result of degeneration extending down the lateral tracts, the reflexes return and become exaggerated, the paralyzed limb becomes spastic, and clonus develops. In from one to four months after the attack the period of late contractures is established, the limbs, as a result, becoming rigid in certain characteristic deformities. Post-hemiplegic motor phenomena, such as rhythmical tremors or irregular movements, choreiform or athetoid, may now appear.

Diagnosis.—During the period of unconsciousness it is necessary to exclude *syncope*, *poisoning*, and *alcoholic intoxication*. If hemiplegia can be detected, nothing more is necessary; but if it cannot, the pale face and weak pulse of syncope, the contracted pupils of opium-poisoning, and the odor of alcohol may aid in distinguishing those conditions. It must not be forgotten, however, that frequently the patient has been given alcoholic stimulants or that intracranial hemorrhage may develop after the patient has taken alcohol. *Uremia* may be yet more difficult to detect, since cerebral hemorrhage is so frequently associated with chronic interstitial nephritis. Convulsive rather than paralytic features usually distinguish such cases, however. *Meningeal hemorrhage* presents similar symptoms, but it usually follows injury after a short interval, and is accompanied by cranial nerve irritation and bilateral convulsions. An attack of *Jacksonian epilepsy* may be due to cortical hemorrhage, but such attacks are common to all lesions of the cerebral motor area. Rarely *hysteria* produces a very exact imitation of intracranial hemorrhage, but the age of the patient and her history are usually significant. *Cerebellar hemorrhage* is sudden in onset, the patient falls, vomits, and is extremely dizzy, while the attack occasions much less disturbance of mind and consciousness than does cerebral hemorrhage.

It is extremely important to both prognosis and treatment that if possible a distinction be made between hemorrhage, the extravasated blood of which may become encapsulated,

and thrombosis, which is a prelude to softening. The following table will aid in this differentiation:

Hemorrhage.	Thrombosis.
Frequent before third year and between fortieth and sixtieth.	Frequent in young adults and in the very old.
Attended by high arterial tension.	Low arterial tension.
Incited by emotions, effort, or shock.	Rarely excitement or effort, except in embolism; rather favored by sleep.
No prodromes.	Prodromes usual.
Sudden, complete stroke.	Less complete.
Coma complete.	Coma slight or wanting.
Face congested.	Face pale.
Respiration stertorous.	Respiration undisturbed.
Pulse slow, full, bounding.	Pulse weak, often frequent.
Hemiplegia, complete at onset.	Often monoplegic, but inclined to extend.
Rapid improvement in motion follows.	Slow improvement, or even extension of the paralysis.
Anesthesia transitory.	Persistent paresthesia.
Persistent aphasia unusual.	Persistent aphasia.
Post-hemiplegic athetosis and chorea common.	Uncommon.
Post-hemiplegic convulsions rare.	Common.

Prognosis.—Intracranial hemorrhage is the result of advanced arterial disease, and hence recurrence is possible. Two-thirds of the patients survive the first attack, but very few a second or third. Prolongation of the coma for a longer period than three days, or the appearance of Cheyne-Stokes respiration, convulsions, pneumonia, or bed sores, presages a fatal termination.

The paralysis, if unimproved within a week, is likely to be permanent. Should improvement begin it is apt to be progressive for the first three months, but after that period only slight further change is to be anticipated.

Treatment.—If the diagnosis of intracranial hemorrhage is positive, active measures to reduce arterial tension may be

adopted: the head raised, an ice-cap placed upon it, and heat applied to the lower extremities. Pressure may be made upon both carotids; or "bleeding into the veins," by applying a tourniquet to prevent the return flow from the lower extremities, may be tried. If there is much arterial excitement, *aconite* or *veratrum viride* should be given; if cerebral congestion is prominent, *belladonna;* and if nephritis coexists, *glonoin.* Marked venous congestion and profound stupor will suggest the administration of *opium.*

As a rule the hemorrhage reaches its maximum within four hours, and thereafter the object of treatment is largely to prevent a recurrence. For this purpose quiet, warmth, cleanliness, and a liquid diet are essential. The bladder should be emptied, and care taken that the inhalation of food or mucus does not set up an aspiration-pneumonia. When coma has passed away, gentle massage and mild faradism may be applied daily to the paralyzed muscles. Contractures should be antagonized by encouraging voluntary control, and for this purpose it is well to have the two sides of the body perform movements simultaneously. *Causticum, baryta carb.* and *sulphur* are recommended as remedies promoting absorption of the clot.

CEREBRAL SOFTENING.

(Embolism; Thrombosis.)

Etiology.—Acute softening of the brain follows the plugging of a cerebral blood vessel by—

1. An *embolus*, generally consisting of small, soft particles of fibrin which have been detached by the blood current from the valves of the heart. Rarely, an embolus may originate in inflammatory or degenerative lesions affecting the vascular system outside of the heart, but as a rule it arises from endocarditis complicating an infectious disease. It is, therefore, most common in the young.

2. A *thrombus*, either (*a*) deposited upon an embolus, or (*b*) developing *in situ* upon the inner coat of a vessel. In the

latter case the vessel-wall is usually degenerated (arteriosclerosis, syphilis), and in addition the blood current is slowed, and consequently the process of coagulation favored, by weakness of the heart's action. It is, therefore, most frequent in the aged.

Pathology.—Because of the deficient anastomosis of the cerebral circulation, occlusion of a vessel cuts off the blood supply from a certain territory. In twenty-four hours this area begins to soften and subsequently it is absorbed, leaving a cicatrix or cyst. If the occlusion is due to an embolus containing infective organisms, local encephalitis and an abscess may result.

An embolus usually enters the brain by one of the carotids, especially the left, and traverses the internal carotid to the left middle cerebral in the fissure of Sylvius; rarely it enters by the vertebral and its posterior cerebral branch. A thrombus affects the middle cerebral and basilar most frequently; it may, however, plug the posterior cerebral, the vertebral, the branches of the circle of Willis, and the basilar at its bifurcation. Occasionally it may involve the sinuses.

Symptoms.—*Embolism* is rarely preceded by prodromes. As a rule, there is a sudden onset of hemiplegia, associated with temporary vertigo, confusion, or loss of consciousness, but little or no coma.

Thrombosis is usually preceded by vertigo, transient aphasia or hemiplegia, numbness of the hand and foot, and drowsiness; in syphilitic cases there may be headaches and cranial nerve palsies. The onset is often rather gradual, hemiplegia developing in the course of several hours and the patient in the meantime becoming comatose. In some cases, however, the onset is so abrupt that it is difficult to exclude hemorrhage.

The acute softening of the brain tissue which ensues may kill within twenty-four hours, but generally the patient survives for several weeks at least. After the acute stage has passed the patient develops the hemiplegic state described as the sequence of intracranial hemorrhage.

Diagnosis.—The distinction between acute softening and

hemorrhage has been considered in connection with the latter disease. Embolism may be distinguished from thrombosis by the age of the patient, the co-existence of heart disease, and the suddenness of the onset.

Prognosis.—In embolism the danger of recurrence is much less than in thrombosis, and the mental condition is better. The prognosis for the hemiplegia, after the chronic stage has been reached, is about the same in embolism, thrombosis, and hemorrhage.

Treatment.—Place the patient at rest in bed with the head raised. Attend to the activity of the heart, kidneys, and bowels. Weakness of the heart may justify the administration of alcoholics or of *digitalis*, preferably in conjunction with *glonoin*. Syphilitic cases should receive *mercury* and the *iodides*. The hemiplegia should be treated in the same manner as that due to hemorrhage.

INTRACRANIAL TUMOR.

Etiology.—The causes of intracranial tumors are as obscure as those of neoplasms elsewhere. Certain varieties (*e. g.*, tubercles, gummata, echinococcus cysts) are readily explained. Heredity may play a part in the production of cancer, gumma, and tubercle; and traumatism may predispose in some cases. The tubercle is frequent in children; otherwise, adult males are most often afflicted.

Pathology.—The most common form is the *tyroma* (tubercle), the encysted caseous masses of which are often multiple. Next in frequency is the *gumma*, not unlike the tubercle in appearance, but, unlike it, capable of retrogression. The *sarcoma* usually occurs as a single encapsulated neoplasm of rapid growth which can easily be enucleated. The *glioma*, a soft, reddish tumor arising from the neuroglia, is infiltrating and destructive in its tendency. It is frequently mixed with sarcoma (*glio-sarcoma*). The *carcinoma*, practically always secondary to a growth elsewhere, develops in the second half of life, and appears as an ill-defined nodular growth which is in-

operable. *Cysts* are usually congenital (*porencephalus*), but are at times the result of hemorrhage or of the presence of parasites (echinococcus, cysticercus). *Aneurisms* springing from any of the cerebral arteries may also give rise to the symptoms of intracranial tumor. Other varieties (fibroma, neuroma, psammoma, leproma, etc.) are rarely found in the brain.

All brain tumors cause a certain amount of destruction in the surrounding tissue; if the meninges are reached, an inflammatory thickening over the growth occurs; and should the return circulation from the ventricles become obstructed, a condition of internal hydrocephalus is added.

Symptoms.—The clinical picture varies according to the situation of the tumor and the rapidity of its growth. As a rule, the symptoms are of two classes, viz.,

General symptoms due to increased intracranial pressure. The more important of these are—

1. *Headache.* In a majority of cases this is early, persistent, and often intolerable. It is generally more intense the nearer the tumor is to the surface, probably through involvement of the meninges. Its location bears no necessary relation to that of the tumor.

2. *Insomnia.* This may be the result of constant pain, but at times it occurs in the absence of the latter.

3. *Vomiting.* Attacks of vomiting of cerebral type, *i. e.*, without nausea or effort, are common, especially if the growth involves the cerebellum.

4. *Vertigo.* Dizziness is particularly apt to accompany tumors in the cerebellar and frontal regions.

5. *Optic neuritis.* Choked disc (papillitis) is present in 80 per cent. of the cases of brain tumor, but it may also be the result of increased intracranial pressure due to other causes (*e. g.*, abscess, meningitis). It is usually bilateral.

6. *Mental impairment.* Mental vigor may be impaired early in the course of the disease, and in the later stages the patient

is apt to sink into stupor or coma. In exceptional cases various psychoses (melancholia, mania, etc.) develop.

Focal symptoms. Various localizing symptoms may occur. They consist of unilateral spasms confined to the face, to a limb, or to a segment of a limb; of aphasia, of monoplegias, of paresthesias, of hemianopia, or of cranial nerve disturbances. They may be associated with or initiate general convulsions of epileptiform type.

Diagnosis.—The existence of a tumor may be indicated by the symptoms enumerated; but it is then necessary to ascertain its location, and this is determined, if at all, by study of the focal symptoms in their relation to cerebral localization. The probable nature of the tumor may often be surmised after consideration of the patient's age, diathesis, and previous illnesses. In childhood, tubercle, glioma and sarcoma are the more common; in adults, the gumma may often be suspected; and in the aged, carcinoma.

Meningitis is more rapid in its development than a brain tumor and the motor symptoms are usually bilateral. *Uremic convulsions* may assume the Jacksonian type, strongly suggesting a focal lesion, but repeated urinary examinations, and especially estimation of the amount of urea excreted, will serve to distinguish the disease.

Prognosis.—In but seven per cent. of brain tumors is operative interference practicable, and only the gumma is amenable to medical treatment. The general prognosis is, therefore, far from favorable. A large majority of cases end fatally within three years.

Treatment.—Syphilitic cases should receive inunctions of *mercury*, and *potassium iodide*, often in enormous doses, should be given internally. If the tumor can be definitely localized, operative removal should be attempted. In the remaining cases only palliation is possible.

ACUTE HEMORRHAGIC ENCEPHALITIS.

Acute hemorrhagic encephalitis is a condition marked by the occurrence of multiple hemorrhagic, softened foci in the brain tissue. It is usually secondary to some infectious disease, notably influenza. The **symptoms** are indefinite, often suggesting meningitis; they consist of headache, vomiting, convulsions, or localized palsies. Sluggish pupils and strabismus are common, and the patient is stuporous and inclined to sink into coma. The **diagnosis** is usually made post mortem, but it is probable that a fair proportion of the cases recover. The **treatment** is that of meningitis.

PURULENT ENCEPHALITIS.

(Brain Abscess.)

Etiology.—Inflammation of the brain when caused by pyogenic organisms eventuates in suppuration. The process may be diffuse or circumscribed. It is invariably secondary, especially to—

1. Suppurative middle ear diseases (40%).
2. Head injury (25%).
3. Pyemia (15%).
4. Septic inflammations in the nose or pharynx, pulmonary tuberculosis, acute infectious diseases, etc., furnish most of the remaining cases.

Pathology.—The pathway of invasion is frequently obscure, but usually the pyogenic organisms are conveyed to the brain through the arteries or venous channels. The frontal and temporal lobes are most frequently attacked. The size of the abscess may vary from that of a pea to that of an orange, and it may be single or multiple. In a majority of cases it is encysted.

Symptoms.—The symptoms are often indefinite, but in many cases three stages are observed, viz.,

1. *The stage of invasion.* An intense and persistent headache, suggesting meningitis, is accompanied by a low, somewhat septic, type of fever, and the occurrence of rigors or sweats occasionally serves to indicate the nature of the process. Mentally the patient is torpid, sometimes delirious. This condition may merge at once into the third stage, or it may be followed by—

2. *A stage of remission*, due to encapsulation of the abscess. The early symptoms may entirely disappear, and for a variable time the patient appears perfectly well. Finally, however,

3. *The stage of paralysis* supervenes. Suddenly the abscess ruptures, infiltrating the capsule, invading the medulla, or tearing through the cortex. The symptoms are those of an apoplectic stroke, sometimes punctuated with focal convulsions; and, as a rule, the patient dies within a few hours. If he should survive the stroke, hemiplegia develops and with it the headache, fever, and other symptoms of the early stage reappear, and death is seldom long delayed.

Diagnosis.—The diagnosis rests largely upon a history of the onset of cerebral and septic symptoms following middle ear disease, head injury, or pyemia. Localizing symptoms are often entirely lacking.

Prognosis.—Grave, and without operative interference, hopeless.

Treatment.—Prophylaxis is most important; suppurations in the ear or nose and injuries about the head should, if possible, be made surgically clean. When the formation of a brain abscess is suspected, the aid of a surgeon should be invoked at once.

SUPERIOR ACUTE POLIOENCEPHALITIS.

(Nuclear Ophthalmoplegia.)

Etiology.—Acute or subacute inflammation, often hemorrhagic, of the central gray matter in the floor of the third ventricle and the aqueduct of Sylvius, involving the upper cranial nerve nuclei, may be due to:

1. Toxic agents, notably alcohol, lead, carbon sulphide, and carbon dioxide.

2. Infectious diseases, especially influenza, pneumonia, diphtheria, and syphilis. The lesion is closely related to that of poliomyelitis anterior, with which it is sometimes associated.

Symptoms.—Premonitory symptoms, such as headache, diplopia, and unsteady gait, or, in alcoholics, stupor or delirium, may precede the attack. As a rule, however, there is a rather sudden onset of paralysis of the upper cranial nerve nuclei on one or both sides. This is indicated by ptosis, internal convergence or fixedness of the eyeballs, and inactive pupils. In consequence the face becomes mask-like and expressionless. These symptoms vary according to the completeness with which the nuclei of the third, fourth, and sixth nerves are involved. Exceptionally they are associated with disturbances of gait, which vary from slight inco-ordination to a reeling suggestive of cerebellar ataxia.

Prognosis.—The prognosis is very grave, especially in children. A majority die, but a few cases become stationary, and others merge into the chronic form.

The **treatment** consists principally of rest and attempts to remove the infective or toxic cause.

SUPERIOR CHRONIC POLIOENCEPHALITIS.

Etiology.—This condition may be:

1. A sequence of the acute form, or a result of the same toxic and infective causes.

2. Secondary to locomotor ataxia, postero-lateral sclerosis, paralytic dementia, or disseminated sclerosis.

Symptoms.—The symptoms are those of the acute form, but are much more gradual in onset. Beginning with a ptosis or squint, the other symptoms are added in the course of perhaps several years. Extension to the bulbar region or even to the cord may ensue.

The **prognosis** is unfavorable. **Treatment** may be directed to syphilis, if the presence of the latter is suspected, or to the toxic cause, if any is discoverable.

INFERIOR POLIOENCEPHALITIS.

(Progressive Bulbar Paralysis; Labio-glosso-laryngeal Paralysis.)

Etiology.—Disease of the nuclei and lower neurons of the seventh, ninth, tenth, eleventh, twelfth, and the motor portion of the fifth cranial nerves may be due to:

1. Extension of the degenerative process attending poliomyelitis (*polioencephalomyelitis*), locomotor ataxia, disseminated sclerosis, syringomyelia, and descending degeneration of the pyramidal tracts following cerebral lesions.

2. Toxemia, induced either by such diseases as syphilis, diphtheria, or chronic nephritis; or by chemicals, such as lead.

Symptoms.—The disease is insidious but progressive. As a rule, the tongue is first affected, and gradually becomes paralyzed and atrophic, lying motionless in the mouth. Soon after this the lips are similarly involved, then the palate, the pharynx, and finally the larynx. Pronunciation becomes thick early in the course of the disease, and speech is gradually lost. The mouth drops open, and from it the saliva drules. Mastication becomes feeble or impossible, fluids regurgitating through the nose Involvement of the pharynx leads to difficulty in deglutition, food being apt to enter the respiratory tract, and the pharyngeal paralysis completely extinguishes the voice. Respiratory and cardiac failure may occasion the fatal termination at any time; but frequently it is precipitated by an aspiration-pneumonia or by asphyxia induced by obstruction of the respiratory passage by a mass of food.

Acute bulbar paralysis may develop in connection with acute myelitis; the symptoms are identical with those of the chronic form, but are rapidly evolved, and death from respiratory failure quickly follows.

Prognosis.—The disease progresses steadily to a fatal termination, usually within a few years. In the acute form death is almost immediate.

Treatment.—In order to avoid asphyxia, the use of the stomach tube or rectal alimentation usually becomes necessary. Faradism may be applied to the paralyzed muscles, and such medicines as *argentum nit.*, *causticum*, *mercury*, *phosphorus*, *plumbum*, and *zinc* may be tried.

INFANTILE CEREBRAL PALSIES.

Etiology.—Paralyses in childhood, the result of lesions of the upper neuron within the cranium, may be divided into:

1. Paralyses of pre-natal onset. These may be due to actual deficiency of brain elements (*agenesis*), to imperfect development of the pyramidal tracts, or to gross cerebral defects (*porencephalia*). Injuries and diseases of the mother are doubtless important factors in the production of these lesions.

2. Birth palsies. Injury to the fetal head by protracted or precipitate labor, or by the use of forceps, is the predominant cause of this class of palsies, most of which are due to meningeal, rarely cerebral, hemorrhage.

3. Postnatal palsies. At this period the lesions are the same as in adults, *i. e.*, hemorrhage, embolism, thrombosis, encephalitis, etc. Acute infectious diseases, convulsions, and traumatism play an important etiologic part.

Symptoms.—A majority of the pre-natal and natal palsies are diplegic in distribution, while those of post-natal origin are hemiplegic. In the diplegic cases the lower limbs are more seriously affected than the upper, and the face often escapes; but mental impairment, often extreme, is usually associated. These children are inactive at birth, and rigidity and contractures may be detected during the early months of life. Athetoid movements and epileptic convulsions develop in many of them. A certain variety of cases, due to premature birth and consequently defective development of the pyramidal tract,

are characterized by spasticity of the lower limbs without mental impairment (*Little's disease*).

In the post-natal cases, the child is usually seized during an acute febrile attack with a convulsion more or less unilateral in its distribution. This is prolonged over several hours or else recurs several times, and during this period the child generally remains unconscious. Later the loss of power on one side is detected. Speech is temporarily lost, but is often regained with surprising rapidity. The paralyzed limbs soon become markedly spastic, the reflexes are exaggerated, and contractures and choreoid or athetoid movements develop. Imbecility and epilepsy are common sequels.

Diagnosis.—The normal electrical reactions, normal trophic condition, and the subsequent spasticity serve to distinguish these cases from those due to cord lesions.

Prognosis.—Complete recovery is impossible. The probability of the avoidance of epilepsy and imbecility is better in the hemiplegic than in the diplegic cases, but in no case can even that be promised.

Treatment.—The management of the apoplectic attack and the paralytic state is the same as that for similar conditions in adults. Surgical intervention may be practicable when the location of a meningeal hemorrhage is fairly localizable; and later, correction of deformities by apparatus or tenotomy is often advisable. In addition, nutrition must be maintained by careful dietetic measures and such constitutional remedies as *aurum*, *baryta carb.*, *calcarea carb.*, *causticum*, *lycopodium*, *mercury*, and *sulphur*. For control of the mental enfeeblement education alone is effective.

PARALYTIC DEMENTIA.

(Paresis; General Paralysis of the Insane.)

Etiology.—Intellectual overwork and strain acting upon a constitution often already weakened by alcoholism and especially by syphilis (70%) are the usual causes of paresis.

Pathology.—The process is a diffuse meningo-encephalitis, in the course of which the brain becomes atrophied, especially its frontal lobe, and the meninges inflamed and thickened. The ganglion cells and nerve fibres are degenerated and in part destroyed.

Symptoms.—The course of the disease is divisable into three periods, viz.:

The Prodomal Period. The patient appears to be neurasthenic, but in addition his conduct becomes "peculiar." He may be guilty of little lapses of memory, making slight errors in speaking or writing, or misusing words; or he may offend the proprieties and show a tendency to alcoholic or sexual excesses. The patient early inclines to exaggerate his self-importance, and plunges into extravagant and impossible schemes, neglecting his legitimate business. Slight motor inco-ordinations, tremor of the facio-lingual muscles, and slurring articulation may be noted at this stage, which merges gradually into—

The Period of Developed Dementia. Speech becomes stammering, the writing is marked by elisions and repetitions of words or syllables, tremor of the face and tongue is marked, the pupils are myotic, unequal, or insensitive, and do not respond to light, and the tendon reflexes are either lost or enormously exaggerated. The patient becomes weak and emaciated, and his mental state is marked by failure of memory and weakened judgment, loss of ethical and esthetic feeling, and emotional excitability; while the delusions which now develop are remarkable for their great exaggeration, whether of exaltation or depression. Most characteristic are the delusions of grandeur; he imagines himself the greatest, the wealthiest, or the strongest man in the world. During this period the patient is seized with apoplectic or convulsive attacks, and the onset of the latter is followed by more rapid and apparent mental failure.

The terminal stage supervenes. Memory fails, speech becomes a jargon, the convulsions increase in frequency, and the

patient becomes helpless and bed-ridden, and finally exhaustion or intercurrent disease induces the fatal termination.

Diagnosis.—In the early stage it is important to exclude neurasthenia and hypochondriasis, and this is usually possible by noting the alteration in character, the speech defect, the tendency to motor inco-ordination, and the pupillary changes. *Cerebro-spinal syphilis* is more sudden in onset, headache, often nocturnal, is more frequent and severe, affections of tongue and speech are wanting, paralytic symptoms appear earlier, the mental symptoms are not characteristic, and delusions of grandeur are lacking.

Prognosis.—The prognosis is hopeless, death usually occurring within five years from the onset and often within three.

Treatment.—The patient should be placed in charge of a trained attendant who will supervise his diet, cleanliness, and general hygiene, and if possible he should be sent away from home. If any suspicion of syphilis exists, an anti-syphilitic treatment should be carried out. Of medicines, *argentum nit.*, *arsenic*, *aurum*, *belladonna*, *hyoscyamus*, *platina*, *secale*, and *stramonium* may be symptomatically useful.

DISSEMINATED CEREBRO-SPINAL SCLEROSIS.

(Insular Sclerosis; Multiple Sclerosis.)

Etiology.—This disease is usually secondary, the principal factors in its production being:

1. Infection. In a large proportion of cases the disease develops soon after one of the acute infectious fevers.

2. Intoxication. Lead, copper, and zinc are said to induce the sclerotic changes. The disease usually appears between the ages of twenty and thirty years. Heredity, traumatism, excesses, etc., may have some predisposing influence.

Pathology.—The irritative cause, whatever its nature, reaches the central nervous system through the blood and there sets up localized degenerative processes. As a result, scattered patches of sclerosis develop irregularly through the white and gray matter of the brain, cord, and spinal nerves.

Symptoms.—In spite of the irregular and scattered lesions, the symptoms are fairly uniform, viz.,

1. *Intention tremor*, *i. e.*, a coarse tremor which disappears while the extremity is at rest, but reappears and augments while movement is going on, is almost pathognomonic of the disease.

2. *Nystagmus* is present in two-thirds of the cases.

3. *Scanning speech*, a peculiar defect in articulation due to separation and accentuation of the syllables of each word, is a common symptom.

4. *Mental enfeeblement* in various degrees can usually be detected.

5. *Amblyopia*, due to optic neuritis or atrophy, is very common. Paretic conditions of the eye muscles and pupillary changes are also frequently present.

6. *Motor weakness* is usually detectable. The gait may be spastic, or it may be of cerebellar type, *i. e.*, staggering. The reflexes are generally exaggerated, and clonus may be discovered.

Apoplectiform attacks, transitory hemiplegias, and vertigo may mark the course of the disease. General sensibility is not affected, and visceral and trophic disturbances are almost unknown.

Diagnosis.—The intention tremor, scanning speech, nystagmus, and irregularities of gait usually render diagnosis easy. *Hysteria* may be distinguished by its stigmata.

Prognosis.—The course of the disease is apt to be prolonged over a period of years. Occasionally it reaches its full development in a few months, and in some cases only a few of the symptoms ever appear (*formes frustes*). It may terminate in dementia, or death may be due to intercurrent disease, apoplectiform attacks, or bulbar involvement.

Treatment.—Attention should be paid to the control of infective processes, general nutrition should be preserved, and internally such remedies as *aurum*, *mercurius*, *plumbum*, and *zinc* may be used.

DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

SPINAL PACHYMENINGITIS.

Etiology.—The inflammation may take place in the loose cellular tissue separating the dura from the bony wall (*external pachymeningitis*). This is always secondary to disease of the vertebræ (Pott's disease), or to inflammatory processes nearby (abscesses, bed sores, etc.). Inflammation of the inner layer of the dura (*internal pachymeningitis*) may be hemorrhagic (hematoma) or hypertrophic, the latter representing simply the thickening and hypertrophy resulting from organization of the effused blood. The process may be extensive or it may be confined to a comparatively small arterial area, especially in the cervical region (*pachymeningitis cervicalis hypertrophica*).

Symptoms.—External Pachymeningitis gives rise to local tenderness over the spine, pain, muscular twitchings, and cutaneous hyperesthesia in the area of distribution of the affected nerves.

Internal Pachymeningitis is slow in onset, beginning with pain over the affected area of the spine and in the peripheral distribution of the corresponding nerves. In the course of months this is followed by loss of power, atrophy, and anesthesia in these areas, together with spastic symptoms, rigidity, and increased reflexes in the lower extremities, due to degeneration of the lateral tracts following compression of the cord.

Diagnosis.—*External pachymeningitis* is distinguished from *myelitis* by the preponderance of pain, spasm, and irritation over paralysis, and by discovery of the causative disease.

Internal pachymeningitis can be distinguished from *amyotrophic lateral sclerosis* by the presence of severe pain in the back and arms, and from *syringomyelia* by the absence of the peculiar sensory changes of that disease. It may be difficult to exclude tumor.

Treatment.—The treatment of either form of pachymenin-

gitis, except in cases of syphilitic origin, is surgical. That associated with Pott's disease is least grave, owing to the success of orthopedic measures in controlling the latter disease. In the other cases surgical intervention, though hazardous, offers almost the only hope. In acute cases the use of *aconite*, *belladonna*, *bryonia*, or *hepar* is often indicated. In cases dependent upon vertebral caries, *calcarea*, *fluoric acid*, *silica*, and *sulphur* may be prescribed. Syphilitic cases require *mercury* or an *iodide*.

SPINAL LEPTOMENINGITIS.

Etiology.—Acute inflammation of the pia mater is due to infection occurring in connection with acute infectious fevers, tuberculosis, syphilis, etc., or to extension of a contiguous inflammation.

Pathology.—The membrane becomes hyperemic and the pia-arachnoid space is filled with a sero-fibrinous or purulent exudate. If recovery follows, the inflammatory exudate is gradually absorbed and a diffuse thickening of the meninges persists, with adhesions between the membranes (chronic meningitis).

Symptoms.—The disease begins abruptly, a sharp chill being followed by intense pain in the back, which radiates around the body and down the limbs. There is tenderness over the spine, and rigidity and spasm of the muscles, causing stiffness of the neck and back and even opisthotonos. Vomiting and convulsions may occur. Medullary involvement may cause dyspnea, Cheyne-Stokes respiration, and cardiac failure. At first the reflexes are increased. Should the patient outlive the acute symptoms, paralysis, anesthesia, atrophy, and contractures will develop in those regions of which the nerve supply is affected.

The chronic form presents the symptoms of the acute in lessened intensity, pain in the back predominating. Complete recovery is rare.

Diagnosis.—The sudden onset of pain in the back, rigidity, hyperesthesia, and irregular temperature and pulse serves to distinguish the disease. *Myelitis* is characterized by paralysis and lack of pain, but a certain degree of myelitis frequently accompanies acute leptomeningitis. *Subdural hemorrhage* presents similar symptoms, but is extremely abrupt in onset, usually following strain or traumatism. *Hemorrhage* into the spinal cord is followed instantly by paralysis, sometimes without pain. *Tetanus* is distinguished by early trismus, fever, paroxysms of spasm, intense hyperesthesia, and a history of traumatism.

Prognosis.—The outlook is grave, some cases terminating in death within two days, others in a week or two; while in those that survive some degree of paralysis usually persists. An acute onset, virulent infection, high temperature, and implication of the upper cord are unfavorable signs.

Treatment.—Place the patient at absolute rest on his side and apply heat or ice bags to the spine. Warm baths are of service in some cases. *Aconite* may be given at the onset, but the developing inflammation soon requires *belladonna*, *bryonia*, or *rhus tox*. *Apis*, *causticum*, *cuprum*, *hypericum*, *oxalic acid*, *secale*, and *veratrum viride*, have also been used with success.

SPINAL HEMORRHAGE.

Etiology.—The hemorrhagic effusion may be extra-meningeal (between the dura and the spinal canal), intra-meningeal (between the dura and the arachnoid), or into the substance of the cord. In the majority of cases it is due to traumatism. In others, rupture of an aneurism, erosion of an artery (caries, carcinoma), or altered blood states (hemophilia, purpura, or those accompanying infectious disease) are responsible for the hemorrhage. Intra-medullary hemorrhage may be secondary to myelitis, meningitis, syringomyelia, etc.

Symptoms.—The symptoms are more or less sudden in onset, and consist of pain in the back, accompanied by numbness or

tingling. If the hemorrhage is meningeal in location, irritative symptoms (hyperesthesia, paresthesia, neuralgic pains, etc.) may be present ; and later, or at the time of onset if the lesion is within the cord, paralysis, usually paraplegia, develops. Consciousness is preserved during the attack. Symptoms of myelitis may ensue.

Diagnosis.—The diagnosis rests upon the sudden onset of the irritative or paralytic phenomena, without loss of consciousness.

Prognosis.—This is unfavorable. Death is not unusual, especially if the cord-substance is involved; and at best some degree of permanent paralysis will remain. Exceptionally a clot, if very small, may be absorbed and recovery ensue.

Treatment.—Place the patient at rest, apply an ice bag to the spine, and administer *aconite*, *arnica*, *hamamelis*, or *veratrum vir.* In general, treat the case as one of intracranial hemorrhage.

ACUTE ASCENDING PARALYSIS.

(Landry's Paralysis.)

Etiology.—This is a rare disease of unknown causation. It appears to follow closely upon various infectious diseases (small-pox, typhoid, influenza, diphtheria, syphilis, etc.), and is probably of toxemic origin.

Pathology.—Myelitic softening of varying extent occurs in the gray matter and the adjacent white fibres of the dorsal and cervical cord, and is accompanied by degeneration in the root fibres and peripheral nerves. The spleen is enlarged and softened, and the lymphatic glands are engorged.

Symptoms.—A sensation of weakness begins in the feet and slowly creeps upward through the legs into the trunk and finally the upper limbs. The parts quickly become paralyzed in the order of involvement, and in the course of two or three days difficult respiration, due to paralysis of the diaphragm, and difficulty in deglutition may appear. In severe cases every voluntary muscle below the face may be paralyzed. In

a few cases the upper limbs are first involved, the paralysis spreading downward. The paralysis may stop at any point and then recede. Sensation is normal. The reflexes are abolished.

Diagnosis.—The diagnosis rests upon:

1. The mode of invasion.

2. The purely motor paralysis.

3. The absence of sensory symptoms and of changes in electrical irritability.

In ordinary *myelitis* the cord functions below a certain level are involved, and sensory symptoms, sphincteric paralysis, and the reaction of degeneration are present. In *acute anterior poliomyelitis* the paralysis reaches its height at once and then tends to recede, and the reaction of degeneration is present.

Prognosis.—The prognosis is unfavorable, the vast majority of cases terminating through bulbar involvement within a few days. Exceptionally, spontaneous arrest is followed by recovery.

Treatment.—Place the patient at absolute rest, nourish him with easily assimilable food, and administer *aluminum*, *cocculus*, *conium*, *gelsemium*, *ledum*, *rhus tox.*, or *secale*.

MYELITIS.

Etiology.—Acute inflammation of the cord substance may be due to:

1. Injuries to the spinal column (fracture, caries, etc.).

2. Injuries to the cord itself (wounds, lacerations, hemorrhages, compression by tumors, etc.).

3. Infectious diseases (syphilis, rarely small-pox, typhoid, septicemia, etc.).

4. Involvement of the cord by extension of a meningeal inflammation.

5. Exposure to cold and wet, and excessive physical exertion act, if at all, by lessening resistance.

Pathology.—The cord becomes reddened and soft, the outline of the gray matter appearing indistinct. The whole thickness of the cord may be involved (*transverse myelitis*), a large vertical section may be inflamed (*diffuse myelitis*), or the lesion may limited to the central gray matter (*central myelitis*). Secondary degenerations ensue in—

(1) The lateral column below the lesion, and

(2) The posterior column and direct cerebellar tract above the lesion.

Symptoms.—The symptoms vary according to the causative lesion and the part of the cord involved. The symptoms common to all localizations are:

1. *Motor paralysis.* Paraplegic weakness develops either suddenly or gradually, and as a result the patient is incapable of actively moving his legs.

2. *Motor irritation.* Muscular twitchings occur in the paretic limbs.

3. *Sensory disturbances.* A band of hyperesthesia surrounds the trunk at the upper level of the cord lesion; it is due to irritation, and is often associated with a "girdle sensation." Below this zone sensation is more or less blunted, and anesthesia, paresthesia, or hyperesthesia may be present.

4. *Changes in the reflexes.* Those reflexes whose spinal centres are involved by the lesion are abolished. Below that level, unless the cord is actually divided, they are at first diminished, but later they become greatly exaggerated and are associated with rigidity and clonus.

5. *Disturbances of Bladder and Rectum.* There may be, early, retention and constipation, but as a rule incontinence of both urine and feces ensues.

6. *Trophic changes.* The muscles innervated from the destroyed cord-segment undergo atrophy and exhibit the reaction of degeneration. The inert limbs may lose in size from disuse, but the electrical reactions are unchanged and the reflexes are increased. The general vasomotor and trophic conditions are disturbed, however, and as a result the paralytic

extremities are cold and cyanotic, the skin is harsh, and bedsores are frequent.

Diagnosis.—The location of the lesion may be determined by the distribution of the paresis and the level of the hyperesthetic zone. *Acute poliomyelitis* is without sensory disturbance; in *meningitis*, spinal tenderness is marked, the girdle pain is absent, and the sphincters are unaffected; in *multiple neuritis* the onset is slow, there is pain along the affected nerves, and the bladder and rectum are not involved; and in *hysteria* the sensory disturbance is characteristic in outline, and no muscular atrophy, bedsores, or sphincteric paralysis is present.

Course and Prognosis.—Acute cases reach their height in a few days, and if death does not occur at once, there is a prolonged stationary period, or a gradual improvement or decline. *Chronic myelitis* differs only in the period required for its development. In any case the prognosis is grave, those that survive the inflammatory period often becoming hopelessly paraplegic, and ultimately perishing from exhaustion or intercurrent disease. In a few cases, however, almost complete recovery takes place. Cervical myelitis is almost invariably fatal, and involvement of the lumbar enlargement is disastrous because of its permanent effects on the sphincters and legs. Acute cystitis and bedsores are unfavorable omens.

Treatment.—Place the patient at rest, and endeavor to prevent bedsores by absolute cleanliness, alcohol baths followed by thorough drying, and frequent changes of position. Catheterization, should it become necessary, must be guarded by strict asepsis. The supervention of cystitis requires that the bladder be washed out. Electricity may be used after acute symptoms have subsided. The principal remedies are *arnica* and *hypericum* in traumatic cases, and *rhus tox.* or *dulcamara* in those following exposure. *Arsenic*, *belladonna*. *cuprum arsen.*, *ergot*, *mercury*, and *strychnia* are also recommended.

ACUTE ANTERIOR POLIOMYELITIS.

(Atrophic Spinal Paralysis; Infantile Spinal Paralysis, etc.)

Etiology.—This acute myelitis of the anterior horns is probably of an infective nature; it occurs mostly in children under ten years of age, and occasionally becomes epidemic.

Pathology.—The sudden paralysis is the result of intense congestion of the anterior cornua. Subsequently softening, usually unilateral, leads to destruction of the multipolar ganglion cells, and so causes a certain degree of permanent paralysis and atrophy. Frequently several foci of myelitis are present at different levels. Ultimately these areas are replaced by connective tissue, and the anterior nerve roots and the muscles themselves become degenerated.

Symptoms.—Without prodromes a child becomes ill and feverish (100°–102°), and in the course of a few hours a flaccid paralysis of one or more of the extremities is noticed. This remains stationary for a few days or several weeks and then gradually declines, but leaves certain muscle-groups permanently paralyzed. The latter soon become atrophied and show the reaction of degeneration. Ultimately the growth of the associated bones may be retarded, and the muscular paralysis permits various distortions to develop. Sensation is unaffected, and the bladder and rectum are not involved.

Diagnosis.—The sudden appearance of the paralysis should render diagnosis easy. *Infantile cerebral paralysis* is almost always ushered in by convulsions limited to one side of the body; convulsions may occur in poliomyelitis as in other febrile diseases of childhood, but they are generalized. The absence of sensory disturbances, bedsores, and sphincteric involvement will exclude *myelitis*. In severe *neuritis* sensory disturbances are present, and the disease is less acute in its onset.

Prognosis.—A fatal termination is rare, and to a certain degree the paralysis will recede, but a portion of the latter is apt to

be permanent. Those muscles which at the end of two weeks show even a slight response to faradism will certainly recover; the others probably will not. Progressive muscular atrophy may develop later in life.

Treatment.—Place the patient at rest, wrap the paralyzed limbs in cotton, and prescribe *belladonna* or *gelsemium*. When the paralysis begins to subside, place a positive electrode over the spine and with the negative electrode apply a mild galvanic current for several minutes daily. Sustain the child's general nutrition with proper food and fresh air, and prescribe medicines according to the constitutional indications present.

CHRONIC ANTERIOR POLIOMYELITIS

(Spinal Form of Progressive Muscular Atrophy)

Etiology.—The cause is practically unknown, although the disease has been attributed to excessive muscular activity, exposure to cold, infectious diseases, and lead poisoning.

Pathology.—The large multipolar cells in the anterior horns of the gray matter in the cord become degenerated and destroyed, and as a result the entire lower motor neuron degenerates and the muscular tissue which it supplies becomes flaccid and undergoes atrophy. Sclerotic changes also occur in the lateral tracts; if sufficiently marked to cause symptoms, the disease becomes amyotrophic lateral sclerosis (*q. v.*)

Symptoms.—The principal manifestation of the disease is a slowly progressive muscular atrophy. Usually it begins in the upper extremities (Aran-Duchenne type), affecting first the muscles of the one hand, the thenar (thumb) and hypothenar (little finger) eminences wasting, and the interossei undergoing atrophy, so that the hand becomes claw-like (*main en griffe*). The other hand becomes involved later. Sometimes the wasting begins about the shoulder first, but in any case it soon involves the other muscles of the back, especially the trapezii and sometimes the respiratory muscles. Less often the atrophy commences in the legs, the gluteal muscles, the

vasti, and the anterior tibials being first attacked. In other cases the atrophy begins in the peronei, later involving the calf and thigh muscles (*peroneal*, *leg*, or *Charcot-Marie type*). The face usually escapes.

As the result of the atrophy, muscular power fails, fibrillary twitchings may be observed, and galvanic and faradic irritability becomes progressively diminished. The reflexes are lost, but the special senses and the sphincters are not affected.

Diagnosis.—The diagnosis rests upon the appearance of wasting in one group of muscles after another, with accompanying loss of power, fibrillary twitchings, changed electrical reactions, and loss of reflexes. *Amytrophic lateral sclerosis* is distinguished by a more rapid course, together with increased reflexes and spasticity. *Muscular dystrophies* begin in childhood, are slower in progress, more frequently involve the lower limbs, the electrical responses are unaltered, and fibrillary twitching is absent. *Neuritis* is more rapid in onset, is usually accompanied by pain, and has no progressive tendency. *Syringomyelia* presents peculiar trophic and sensory phenomena.

Prognosis.—The course of the disease is prolonged, and it is rarely checked. In the course of years bulbar involvement or invasion of the respiratory muscles may precipitate a fatal termination.

Treatment.—Moderate exercise in the open air and good food are necessary. Galvanism may be applied to the spine daily, and such remedies as *arnica*, *arsenic*, *cuprum*, *gelsemium*, *mercurius*, *phosphorus*, *physostigma*, and *sulphur* may be prescribed.

Strychine nit., in hypodermatic doses of gr. $\frac{1}{100}$, increased to gr. $\frac{1}{40}$, daily, is said to arrest the progress of the disease.

AMYOTROPHIC LATERAL SCLEROSIS.

Etiology.—The cause is unknown.

Pathology.—Degeneration of the pyramidal tracts in the cord (upper motor neuron) is associated with destruction of the ganglion cells in the anterior horns and medulla (lower

motor neuron) with consequent atrophy of the muscles dependent upon the latter. Other portions of the cord are sometimes slightly involved.

Symptoms.—The disease is characterized by a combination of the symptoms of progressive muscular atrophy and spastic paraplegia, viz.,

1. *Progressive muscular wasting* and a corresponding degree of motor weakness. As a rule these atrophic symptoms appear first in the upper extremities, involving the muscles in the order described as usual in cases of chronic anterior poliomyelitis. Fibrillation and diminished electrical irritability can be detected.

2. *Rigidity*, *increased reflexes*, and sometimes contractures. These spastic symptoms usually appear in the lower extremities, giving rise to a characteristic spastic gait. The sphincters are unaffected and cutaneous sensibility is normal.

Diagnosis.—The exaggerated reflexes and spastic phenomena serve to distinguish the disease from chronic anterior poliomyelitis.

Prognosis.—Hopeless. Death will probably result from exhaustion or bulbar involvement within two or three years.

Treatment.—Rest, massage, and such remedies as *argentum nit.*, *arsenic*, *lathyrus*, *plumbum*, and *strychnine*.

SPASTIC PARAPLEGIA.

(Lateral Sclerosis; Spastic Spinal Paralysis.)

Etiology.—Degeneration of the pyramidal tracts may follow a lesion of the upper neuron situated at any point between the motor cortex and the spinal termination of the axon. Occurring bilaterally the condition may be secondary, therefore, to any transverse lesion of the cord above the lumbar enlargement. In rare cases it may be primary.

Symptoms.—The condition is of very gradual onset, the principal symptoms being—

1. *Weakness of the lower extremities*, which gradually progresses to total disability. The arms are rarely involved.

2. *Rigidity in the muscles of the lower extremities.* This spasticity corresponds to the degree of paralysis. The tendon reflexes are greatly exaggerated and ankle clonus can be obtained.

The "spastic gait" is characteristic; the patient moves his legs as a whole without bending them, and the toes scrape the ground. In children there may be "cross-legged progression," because of spasm of the abductors. There is little or no sensory disturbance or muscular atrophy, the electrical reactions are normal, and the sphincters are seldom affected.

A *hereditary spastic paraplegia* has been described, notably by Bayley, who traced a series of cases through five generations. Spasticity and increased reflexes were the essential phenomena.

Diagnosis.—The association of spasm with motor paralysis furnishes a diagnosis of the condition, but only when a careful, persistent search for a causal lesion has been in vain can the disease be considered primary.

Hysterical paraplegia usually presents some rigidity, but it is not true spasticity and the knee jerk is not so markedly exaggerated, nor can clonus be obtained. *Ataxic paraplegia* is distinguished by inco-ordination; *amyotrophic lateral sclerosis*, by muscular atrophy.

Prognosis.—Recovery is impossible and improvement is exceptional.

Treatment.—Rest, massage. and the administration of such medicines as *argentum*, *lathyrus*, *nux vomica*, *phosphorus*, *strychnine*, and *zinc*.

ATAXIC PARAPLEGIA.

(Combined Sclerosis; Postero-lateral Sclerosis; Progressive Spastic Ataxia.)

Etiology.—Simultaneous degeneration of the lateral and posterior columns is usually secondary, especially to—

1. Locomotor ataxia, an associated myelitis involving the lateral tracts.

2. Paralytic dementia, by a similar extension.

3. Diffuse myelitis, giving rise to ascending degeneration of the posterior and descending degeneration of the lateral tracts.

4. Leptomeningitis, an associated myelitis involving the periphery of the cord.

5. Vascular lesions, sclerosis of the lateral and posterior tracts being secondary.

6. Embryonic deficiency.

Exposure, traumatism and syphilis are sometimes credited with inducing the disease.

Pathology.—The process begins in the lumbar region, and the posterior and lateral columns are both affected, but with unequal intensity. The posterior root zone usually escapes.

Symptoms.—The symptoms are, roughly speaking, those of spastic paraplegia and ataxia combined. The esssential characteristics of the disease are:

1. *Ataxia*, especially of the lower extremities.

2. *Gradual loss of power in the lower extremities* with *spasticity* and *increased reflexes*. Some cases show a preponderance of spasticity, while others incline more to the tabetic type, with loss of sexual power, sphincteric paresis, girdle sensation, dull aching, and more rarely lightning pains, Argyll-Robertson pupil, and even loss of the knee jerk.

Diagnosis.—The diagnosis rests upon the association of ataxia with muscular weakness and spasticity.

Prognosis.—The disease is slowly progressive, death occurring eventually, as in other spinal degenerations, as the result of cystitis, bed sores, or intercurrent disease.

Treatment.—That of locomotor ataxia.

FRIEDREICH'S ATAXIA.

(Hereditary Ataxia.)

Etiology.—This degenerative disease of the posterior and lateral columns, which is thereby closely related to ataxic paraplegia and locomotor ataxia, usually attacks several children of one family, appearing before adolescence. In some cases, generally those appearing after puberty, the cerebellum is involved (*hereditary cerebellar ataxia*).

Symptoms.—The principal symptoms are:

1. *Ataxia.* The child is very unsteady on its feet, stumbling, staggering, and reeling. The movements of the arms become unsteady and jerky, and tremor and choreiform movements may be noted. Ultimately even the head becomes involved.

2. *Speech disturbance.* Inco-ordination renders speech jerky or hesitating, with elision of certain syllables; and the pitch changes suddenly at times.

3. *Nystagmus.* Frequently this oscillation develops only when the eyes are directed laterally or upward.

4. *Changes in the deep reflexes.* In cases of the spinal type the knee jerk is lost early; in the rarer cases of cerebellar type, the deep reflexes are exaggerated and clonus may be associated.

5. Muscular strength is but slightly reduced. Late in the disease club-foot and lateral spinal curvature may develop.

Diagnosis.—The hereditary character of the disease and its onset in early life are usually distinctive. *Ataxic paraplegia* lacks the nystagmus and defective articulation. *Disseminated sclerosis* may be distinguished by its intention tremor, its lack of static ataxia, its scanning speech, and its spastic features.

Prognosis.—The disease is incurable but runs a protracted course, extending over many years.

Treatment.—This is very unsatisfactory. Galvanism, spinal stretching, and prescribed exercises may be tried.

LOCOMOTOR ATAXIA.

(Tabes Dorsalis; Sclerosis of the Posterior Columns.)

Etiology.—The principal and perhaps the only factor in the production of locomotor ataxia is syphilis (90%), the disease usually appearing from five to fifteen years after the initial lesion. As a natural corollary, it is a disease principally of adult males.

Pathology.—The syphilitic toxin primarily affects the cell-bodies of the afferent sensory fibres, with ensuing degeneration in the peripheral nerves, the posterior roots, and the posterior columns of the cord, and with the latter meningitis may be associated. Similar changes may occur in the medulla and the brain, the latter constituting paralytic dementia. These degenerative changes are sclerotic in nature and irreparable (parasyphilitic).

Symptoms.—The more important symptoms of tabes are as follows:

1. *Disturbances due to loss of the muscular sense.* As a result of this the patient cannot determine the position of his limbs, and is unable to co-ordinate his movements. Early he presents *static ataxia* (Romberg's sign), swaying or falling when he attempts to stand with closed eyes. Later the *ataxic gait* develops; the patient's movements are unsteady, the foot is raised too high, thrust forward too far, and comes down flat with a slap. In time it becomes necessary for him to use one or two canes and to watch his feet while he walks, and ultimately walking, even with these aids, is impossible. The upper extremities may become markedly ataxic, the patient being unable to touch his nose, ear, or other object when his eyes are closed, and the ordinary movements of the arms becoming very inexact. *Involuntary movements*, consisting of sudden jerks of the extremities, are sometimes observed. *Palsies*, due to involvement of the anterior horn or to vascular changes in the cerebellum or cord, appear in a minority of the cases. They are usually mild and transitory.

2. *Sensory symptoms.* The most important sensory disturbances are the *lightning* (*fulgurant*) *pains*, darting or stabbing in character and lasting but a second or two. Frequently they are accompanied by more or less constant burning or tingling sensations. Pain is also associated with the various tabetic crises. Many patients complain of a sensation as of a tight belt about the trunk (*girdle sensation*), and in a majority of patients a band of anesthesia can be detected about the chest (*tabetic cuirass*). Either *analgesia* or *hyperalgesia* is apt to be present, and is usually of rather symmetrical distribution. Sensation is sometimes delayed, and even the syringomyelic dissociation of cutaneous sensibility may be encountered.

3. *Reflexes.* The knee jerks are diminished, unequal or lost (*Westphal's sign*) early, and the ankle jerk fails at the same time. Later the iris reflex to light is abolished, accommodation remaining unaffected (*Argyll-Robertson pupil*). The organic reflexes may be involved, with resulting *loss of control of the sphincters;* and there is loss of sexual appetite, with more or less impotence.

4. *Visual Disturbances.* In addition to the loss of the light reflex, many other ocular changes may occur in locomotor ataxia, notably *atrophy of the optic nerve.* Pupillary disturbances, ptosis, or squint may develop.

5. *Visceral Symptoms.* Paroxysmal attacks of disturbed function and pain (*tabetic crises*) are very common. They may affect the stomach, causing vomiting and prostration; the intestine, causing diarrhea or constipation and often tenesmus; the urinary apparatus, causing intense colicky pains with great desire but inability to urinate; or the larynx, causing dyspnea and cough, and even cyanosis.

6. *Trophic disorders.* The nutritional disturbance may lead to striking dystrophies, notably spontaneous fractures and tabetic *arthropathies* (*Charcot's joints.*) The latter are characterized by the rapid onset of a painless swelling, without redness or puffiness, about a joint. This may quickly subside, but in severe cases it becomes permanently localized in a

globular form about the joint. There is a tendency to the recurrence of these attacks, with ultimate disintegration of the articular structures, as a result of which the old tabetic joint may be a mere bag of bone fragments. A *perforating painless ulcer*, usually in the foot, is not uncommon; and many other less typical trophic changes may be present.

Course.—The onset of tabes is insidious and its course slowly progressive, but three stages are described, viz.,

1. *The Pre-ataxic Stage.*—The chief symptoms at this period are lightning pains and loss of the knee jerk. The gait may be unaltered, but often there is static ataxia and an Argyll-Robertson pupil.

2. *The Ataxic Stage.*—This cannot be sharply distinguished from the first stage, but is marked by the development of an ataxic gait. The early symptoms persist and become intensified, visceral crises and arthropathies appear, and sphincteric control is lost.

3. *The Paralytic or Terminal Stage.*—The patient becomes unable to "use his legs" because of increasing ataxia, and is practically helpless. The previous symptoms continue and may be more fully developed. Cystitis, bulbar involvement, or some intercurrent disease may induce a fatal termination at any time.

Diagnosis.—The diagnosis of a fully developed case, with lightning pains, loss of knee jerks, ataxia, and Argyll-Robertson pupil, presents no difficulties. In the early stage the condition may be more obscure, but loss of knee jerk together with either static ataxia or lightning pains justifies a conclusion provided neuritis (*q. v.*) and anterior poliomyelitis can be excluded. In certain cases the disease begins in the cervical region, and in consequence it is marked by little inco-ordination or static ataxia and for a time the knee jerks may be retained. In such a case *syringomyelia* may be suspected, but as a rule the latter presents dissociation of cutaneous sensibility, local atrophies early in the disease, persistently increased knee jerks, and absence of inco-ordination. *Para-*

plegias are usually accompanied by exaggerated reflexes and often clonus.

Prognosis.—Recovery is impossible, but in some cases the progress of the disease may be checked. Optic atrophy is often followed by arrest of the ataxic conditions.

Treatment.—1. Control any active syphilitic process. As a rule the luetic manifestations are past, but if they are still present, *mercury* and the *iodides* should be given.

2. Preserve the general health. This implies attention to hygiene and diet.

3. Increase the nutrition of the cord by daily suspension, using the Sayre apparatus, or, better, seat the patient on a table or on the floor and forcibly depress the head toward the knees and preserve this cord-stretching attitude for two minutes.

4. Systematic exercises aid in the preservation of muscular control. The patient should be encouraged to perform certain prescribed definite movements for a few minutes each day.

5. Medicines. *Argentum nit.* is the remedy most often indicated in locomotor ataxia. *Alumina*, *aurum*, *phosphorus*, *picric acid*, *secale*, *zinc*, and *zinc phosphide* are useful at times, and a host of other drugs may be suggested by intercurrent symptoms. The severity of the lightning pains may sometimes justify the use of analgesics (*antipyrine* or *acetanilid*, gr. v–x, rarely *morphia*, gr. $\frac{1}{8}$–$\frac{1}{4}$), but great caution must be observed in the repetition of such medicines.

SYRINGOMYELIA.

Etiology.—Syringomyelia appears to affect especially men who have been accustomed to hard labor. Exposure, traumatism, and the infectious diseases have been suggested as causes, but their relationship is purely conjectural.

Pathology.—A gliomatous infiltration takes place around the central canal or in the gray horns of the cord, and subsequently undergoes degeneration, forming cystic cavities.

Rarely a central myelitis or hemorrhage may lead to a similar excavation. The cavity lies in the gray matter and may be in the position of the central canal. Secondary degeneration may appear in the anterior or posterior columns or horns.

Symptoms.—The symptoms may, according to the extent of the lesion, embrace changes in all the cord functions. The disease is of slow onset, beginning in early adult life, and is usually marked by the following symptoms:

1. The *temperature and pain senses are lost*, while the *tactile and muscular senses are preserved* (the *syringomyelic dissociation*).

2. *Muscular atrophy*, varying in its location according to the portion of the cord diseased, is usually present. It is most frequent in the upper extremities, and is accompanied by fibrillary twitchings and the reaction of degeneration.

3. *Spasticity* of the muscles develops when the lateral tract becomes involved.

4. *Trophic phenomena*, such as glossy skin, hypertrophic nails, paronychia, herpetic and bullous eruptions, etc., are frequently seen. *Arthropathies* are almost invariably present, affecting the spine (scoliosis, angular deformities) and the articulations of the upper extremities by preference.

The special senses and the sphincters are normal.

Morvan's disease is a clinical variety of syringomyelia in which the latter is probably complicated by peripheral neuritis. The sensory dissociation, especially in the hands and arms, is accompanied by atrophy and paresis. The fingers are affected by painless whitlows, and necrosis of the phalanges may lead to great deformity.

Diagnosis.—This rests upon discovery of the loss of the pain and temperature senses and the preservation of tactile and muscular sense, in association with muscular wasting. *Hypertrophic pachymeningitis* may resemble syringomyelia, but the tactile sense is usually lost, pain is greater, and the syringomyelic dissociation is not present. This and other permanent

cord irritations may bear an etiologic relationship to syringomyelia, however.

Prognosis.—The disease is incurable, but the course may be prolonged over fifteen or twenty years.

Treatment.—This is limited to the treatment of symptoms as they arise.

CAISSON DISEASE.

(Diver's Paralysis).

Etiology.—Persons working in caissons and diving bells under increased atmospheric pressure may, on returning to the surface, be seized with pains and paralytic symptoms. The cause is presumably circulatory, but its exact nature is unknown.

Symptoms.—Within half an hour after returning to the surface the patient is seized with pains in the ears and joints, dizziness, headache and nausea; and this is suddenly followed by hemiplegia or paraplegia. A few become comatose and die; but the majority convalesce in a few days and others in the course of some weeks.

Treatment.—By allowing five minutes to each "lock," by means of which the air-pressure is progressively reduced, the disease can be avoided. Upon the appearance of symptoms the patient should be placed at absolute rest, and such remedies as *arnica*, *belladonna*, *bryonia*, *causticum*, *nux vomica*, and *rhus tox.* administered. *Morphine* may be sometimes required for relief of the agonizing pain.

DISEASES OF THE PERIPHERAL NERVES.

NEURITIS.

Etiology.—Inflammation of a peripheral nerve may be due to:

1. Traumatism (compression, contusion, or wounds).

2. Extension from a neighboring inflammation (arthritis, meningitis, etc.).

3. Toxemia, the result of infectious diseases (diphtheria,

syphilis, malaria, etc.), of poisoning from extrinsic sources (alcohol, arsenic, lead, mercury, etc.), or of auto-intoxication (diabetes, gout, etc.).

4. Exposure to cold or dampness.

Pathology.—The inflammation is usually confined to the nerve sheath or septa, but ultimately a connective tissue hyperplasia may ensue, which by compression destroys the nerve itself. Neuritis may be localized to a single nerve or area (*simple neuritis*) or widespread, involving many nerves (*multiple neuritis*).

Symptoms.—Simple neuritis of a sensory nerve is accompanied by pain and tenderness along its course, together with various trophic changes (redness and edema of the skin, herpes, etc). Later, anesthesia succeeds the hyperesthesia. Should inflammation involve a motor nerve, muscular twitchings, impaired motion, and paralysis are apt to result; and later atrophy, contractures, and the reaction of degeneration may supervene.

Multiple Neuritis (*polyneuritis*) is always of toxic origin, and it involves many nerves simultaneously or in rapid succession. The onset may be gradual, or it may be acute and accompanied by fever and febrile symptoms. The typical manifestations vary in their relative intensity, but in some degree they always include:

1. *Motor weakness.* The motor loss is usually more marked in the lower limbs; there is foot-drop, and a characteristic gait in which the patient bends the thigh in order to raise the foot from the ground and the leg is then thrown forward like a flail ("steppage"). The dangling foot wears the shoe at the toe and frequently scrapes up the dirt of the street. In the upper extremities wrist-drop may be conspicuous, and in some cases a claw-hand. Rarely the thigh, trunk, shoulder, and face muscles are involved. Tremor, fibrillation, atrophy, and contractures may be evolved in the course of the disease. The reflexes are diminished or abolished, and the electrical tests usually show the reaction of degeneration in the diseased nerves.

2. *Sensory derangements.*—As a rule the onset is attended by various paresthesias, such as numbness, heat, cold, etc.; and varying degrees of hyperesthesia and anesthesia may also be present. The muscles are apt to become very sensitive as the disease progresses, the slightest pressure causing acute pain; and the patient may suffer with crises of intense agony. The loss of the muscular sense sometimes leads to decided ataxia.

Various trophic changes occur, especially in the skin, and mental symptoms, such as irritability, depression, forgetfulness, or delirium, are sometimes present.

Special Forms.—The *alcoholic* form is most common; it is characterized by a preponderance of sensory over motor symptoms, and the latter are confined principally to the lower limbs. The *lead* palsy is usually preceded by colics and constipation, and the upper extremities are almost exclusively affected. *Erythromelalgia* is a variety of multiple neuritis which especially affects the plantar nerves and is associated with burning pain, redness, and swelling in the feet. *Beri-beri* or *kakké* is a polyneuritis, possibly due to a specific organism, which is endemic in the Orient.

Diagnosis.—The diagnosis of simple neuritis rests principally upon the localization of pain, weakness, paresthesia, and anesthesia to the area supplied by a certain nerve. The diagnosis of multiple neuritis is rarely difficult. It may be necessary to exclude *poliomyelitis*, which is most frequent in children, abrupt in onset, is not usually symmetrical, has fewer sensory symptoms, and begins to improve almost at once but rarely recovers completely. *Myelitis* is distinguished by its girdle pain, paraplegia, anesthesia, and preservation of the electrical responses and deep reflexes. Multiple neuritis sometimes closely resembles *locomotor ataxia* and is referred to as pseudotabes. The two diseases may be distinguished by reference to the following table:

Locomotor Ataxia.	Multiple Neuritis.
Girdle pain, lightning pains.	No girdle pains, rarely lightning pains.
Nerve trunks insensitive.	Nerve trunks tender.
Atrophy and reaction of denenera-tion absent.	Present.
Characteristic gait, due to inco-ordi-nation.	Gait due to muscular paresis.
Strikes heel first.	Drags toe on ground, "steppage."
No vasomotor change.	Edema, discoloration, etc.
Argyll-Robertson pupil.	Absent.
Paralysis of sphincters common.	Unusual.
Perforating ulcers, arthropathies.	None.

The discovery of the toxic cause of multiple neuritis is important, yet often difficult. Careful investigation as to alcohol, lead, arsenic, diphtheria, etc., usually reveals the probable origin, however.

Prognosis.—This must be governed by the intensity of the symptoms and the extent of degeneration revealed by the electrical reactions. In simple neuritis it is generally favorable, although in severe cases recovery may be delayed for months. In multiple neuritis it is usually good, provided the cause can be removed.

Treatment.—The affected area, whether a single extremity or the entire body, should be put at rest. The arm may be carried in a sling. If possible the cause must be removed. The diet should be very plain and non-stimulating. Hot applications (hot cloths, hot water bags, or the Japanese fire-box) serve to relieve the pain. Galvanism, a mild uninterrupted current, may be used for five or ten minutes daily, and massage is a useful adjunct. If the pain is severe, *belladonna* is usually indicated, although in severe cases of multiple neuritis with burning pain *arsenic* is frequently preferable. *Rhus tox.* is indicated in cases due to exposure, *gelsemium* and *causticum* in cases presenting marked paralytic symptoms, and *argentum nit.* when ataxia is present. *Cimicifuga*, *nux vomica*, or *strychnine* may be given in alcoholic cases.

NEURALGIA.

Etiology.—Nerve pain is merely a symptom. It may be due to any condition which produces irritation of a nerve. The more important predisposing causes are:

1. *Heredity.* Neuralgia is particularly common in the victims of neuropathic heredity.

2. *Age.* It is more frequent between the thirtieth and fiftieth years of life.

3. *Sex.* Women appear to suffer from it more often than men.

4. *Occupation.* Painters and workers in metal are very liable to neuralgia.

5. *General physical condition.* Any impairment in health predisposes to neuralgia.

Among the exciting causes are:

1. Exposure to cold or wet.
2. Traumatism.
3. Irritation (thermal, chemical, or mechanical).
4. Toxic states (gout, infectious diseases, intoxications, etc.).
5. Neuromata.

Symptoms.—The characteristic neuralgic pain is unilateral, inconstant, and usually paroxysmal. It is described as darting, stabbing, tearing, etc., is deep-seated, and is often accompanied by cutaneous hyperesthesia. Between pains tenderness can be detected at certain points where the nerve is superficial, overlies bone, or is inclosed by rigid tissue ("tender points" of Valleix). The pain may be accompanied by muscular spasm (*e. g.*, tic douloureux), and occasionally local vasomotor symptoms (flushing, etc.), or trophic changes (erythema, herpes, pigmentation, etc.) may be noted.

Varieties.—Among the more important varieties of neuralgia, classed according to location, are the tri-facial, cervico-occipital, cervico-brachial, dorso-intercostal, intercostal, lumbo-

abdominal, spinal, sacral, sciatic, crural, metatarsalgia, etc. Visceral forms (*e. g.*, gastralgia, enteralgia, etc.) are also noted.

Diagnosis.—The diagnosis of neuralgia rests upon the unilateral pain, its limitation to the area supplied by certain nerve trunks, its paroxysmal character, and the absence of symptoms pointing to other conditions. In *neuritis* the pain is more constant, the functions of the nerve are interfered with, anesthesia and paralysis ensue, and the nerve trunk is more tender.

Treatment.—Ascertain the cause, local or constitutional, and if possible remove it. Rest, nourishing food, and proper hygiene are usually prerequisites to successful treatment of the more chronic cases. *Belladonna* is useful in acute neuralgias of sudden onset and attended by congestive symptoms. *Mezereum* is particularly valuable in facial neuralgias resulting from the irritation from decayed teeth, *arsenic* and *cedron* in neuralgias of malarial origin, *cimicifuga* and *bryonia* in those of rheumatic nature, and *colchium* and *rhus tox.* in cases associated with a gouty state. The intense pain may often be relieved by the application of dry heat, but at times it is necessary to give *acetanilid* or *antipyrine*, gr. v–x, rarely *morphia*, in order to relieve the suffering. In obstinate cases uncontrolled by general and medicinal measures it may be advisable to resort to stretching or resection of the nerve.

SYPHILIS OF THE NERVOUS SYSTEM.

Varieties.—Syphilis gives rise to an almost endless variety of lesions of the nervous system. These may be divided into:

1. Lesions due to direct microbic action (*specific lesions*).

2. Lesions sequential to syphilis, not neoplastic in character, and due to some toxic agent (*parasyphilitic lesions*).

A systematic tabulation of the lesions (Church) follows:

- Syphilis of the general nervous system.
  - Active specific lesions.
    - Cerebro-spinal syphilis, acquired and hereditary (early and tardy).
      - Cerebral.
        - Meningitis.
        - Cerebritis.
        - Arteritis.
        - Neuritis.
        - Mania.
        - Melancholia.
        - Pseudoparesis.
      - Spinal.
        - Meningomyelitis.
        - Myelitis.
        - Spinal paraplegia.
    - Syphilis of spinal nerves.
  - Parasyphilitic Disease.
    - Acquired.
      - Locomotor ataxia.
      - Paralytic dementia.
      - Neurasthenia.
      - Hysteria.
      - Epilepsy.
      - Neuralgia.
    - Hereditary.
      - Juvenile tabes and paresis.
      - Infantilism.
      - Hydrocephalus.
      - Cerebral or spinal agenesis.

Diagnosis.—CEREBRAL SYPHILIS presents some features which are so characteristic that even without a history of previous infection the diagnosis is not difficult. The *development* of brain-syphilis is apt to be marked by a series of advances, each followed by a partial remission; it is almost never abrupt in onset. *Nocturnal headache*, often atrociously severe, and frequently associated with cranial nerve symptoms (diplopia, ptosis, pupillary changes, optic nerve disturbances) is well-nigh pathognomonic. *Epileptoid attacks* appearing after the thirtieth year of life, and *apoplectiform seizures* occurring before the age of fifty, afford strong presumptive evidence of syphilis. A *multiplicity of lesions*, *e. g.*, brain and cord symptoms in the same patient, should arouse suspicion. Finally, the *therapeutic test* (large doses of the iodides), if followed by improvement within a week, strongly sustains the diagnosis of syphilis. It must not be forgotten, however, that the symptoms of other organic brain diseases, even those due to a neoplasm, may be somewhat ameliorated by the administration of iodides.

SPINAL SYPHILIS affords far greater diagnostic difficulty. It has no characteristic premonitory symptoms, and its distinction must often rest upon a history of infection or upon associated cerebral symptoms of a suggestive sort. The irregular advance and retreat of the symptoms is somewhat significant.

Prognosis.—The prognosis of cerebro-spinal syphilis is by no means as favorable as is popularly supposed, less than one-

fourth of the cases recovering completely. This is, of course, because of the disintegration and destruction of the tissues secondary to the specific lesion; and consequently early discovery and energetic treatment of the latter becomes doubly imperative.

Treatment.—Administer at first twenty drops of a saturated watery solution of sodium iodide in a glassful of water or milk three times a day, after meals. Increase this dose by ten drops daily until, in critical cases, 100 or even 200 drops are given at a dose. In the meantime it is often advisable to give mercury by inunction; ʒss–ʒj of the ointment should be applied daily to different parts of the body, as directed in the general treatment of syphilis (page 108). After a week or two omit the mercury and continue the iodide alone for a week; then stop the latter, and return to the mercurial inunctions for a week; and so continue to alternate the remedies. The dose should be decreased as the symptoms yield, but must be continued for six months; and for five years thereafter the patient should undergo an annual course of six months' specific treatment.

THE NEUROSES.

(Functional Nervous Diseases, *i. e*, Diseases of Undetermined Pathogenesis.)

HYSTERIA.

Etiology.—Hysteria is a psychoneurosis the exact nature of which remains undetermined, but its manifestations are undoubtedly due to the control of the body by ideas. Among the important predisposing factors are:

1. *Sex.* A decided majority of hysterical patients are females.

2. *Heredity.* A hereditary neurotic taint is the most potent factor in the production of hysteria. Other neuroses and diatheses, notably gout and tuberculosis, are important antecedents of hysteria, as of other forms of degeneracy.

3. *Age.* The period of puberty and adolescence furnishes a majority of the cases of hysteria. As life advances the frequency of the disease lessens, and after the age of 45 it is rare.

The exciting cause of the hysterical manifestations is almost invariably an emotional disturbance, due to mental or moral strain (overwork, worry, grief, etc.), or to shock, either psychical or physical (fright, traumatism). The disease may become epidemic in small, closely-associated communities, such as schools, nurseries, and remote villages, as the result of imitation, a so-called "moral contagion."

Symptoms.—The manifestations of hysteria are divisable into two groups, one of which is persistent so long as the disease lasts—the stigmata; and a second group consisting of symptoms which appear incidentally and are often transitory—the paroxysmal phenomena.

A. **Stigmata.** 1. SENSORY STIGMATA. These disorders may be negative, *i. e.*, anesthesia, or positive, *i. e.*, hyperesthesia.

Hysterical anesthesia may affect sensation in all its varieties and distributions. Cutaneous insensibility may be so absolute that neither pinching, pricking, nor the application of hot or cold substances to the skin elicits the slightest response; and the mucous membranes of the mouth and throat and other orifices are often equally insensitive. The deeper tissues are also frequently anesthetic, so that muscles, ligaments, and nerve trunks may be twisted or pierced without evoking the slightest sensation. The muscular sense may be so completely abolished that the patient when blind-folded has no knowledge of the position of a limb, nor can she recognize pressure or fatigue.

The *special senses* may be equally affected. Smell and taste may be perverted, diminished, or abolished, the acuity of hearing may be lessened, and vision may be modified or abolished. More commonly the visual disturbance consists of—

(*a*) Contraction of the visual field. This is usually concentric and often bilateral.

(*b*) Difficulty in color perception (*dyschromatopsia*). In health blue has the largest field, followed by yellow, orange, red, green, and violet. In hysteria, and also in neurasthenia, these fields may be contracted, and, more important, their order is changed, the red field exceeding the blue; and should the color fields become extinguished, red is the last to disappear. All objects then appear grayish in color (*achromatopsia*).

(*c*) Errors of accommodation. Near vision may be rendered faulty, or monocular diplopia, macropsia, or micropsia may be present. The patient may aver that she sees nothing with one eye, yet perceives two images when a prism is placed before either eye, indicating that she sees without knowing it. A characteristic symptom is "crossed amblyopia," one eye, particularly that on the side which is paralyzed or anesthetic, seeing poorly, while the other is less affected.

The *distribution of hysterical anesthesia* is characteristic. Rarely the entire skin and membranes are affected; as a rule it is:

(1) Limited to one-half the body, especially the left (hysterical hemianesthesia).

(2) To definite areas, usually about an extremity on which may be outlined the position of a sleeve, glove, sock, or stocking. In any case the outlines have no relation to the area of distribution of the nerve trunks.

(3) Discrete islets of anesthesia may be present, minute search being necessary for their discovery.

The *peculiarities of hysterical anesthesia* are:

(*a*) The area does not conform to the distribution of the cutaneous nerves or to the spinal segments.

(*b*) The pupillary and tendon reflexes are not modified as in organic lesions.

(*c*) The areas are movable, enlarging, shifting, or at times disappearing.

(*d*) The patient is usually ignorant of the existence of these anesthetic areas.

Hysterical hyperesthesia is common. There may be a fixed boring pain in one spot, usually the temple (clavus), or there may be pain in the back, the breasts, in the pit of the stomach, or in certain joints. Tenderness is even more common. If such a hyperesthetic area has been associated with some mental storm, pressure upon it, by reviving the memory, may provoke a hysterical seizure (hysterogenic zone).

2. Motor Stigmata. These stigmata must be carefully distinguished from the paroxysmal motor phenomena. They consist simply of slight retardation, inco-ordination, or weakness of intentional movements, together with a tendency to rigidities or contractures.

3. Mental Stigmata. These stigmata consist of:

(*a*) *Amnesia* (loss of memory), the patient's lack of mental concentration leading to forgetfulness and consequently uncertain and contradictory statements, or to the loss of certain motor images, such as those for walking, writing, or speech, with astasia, agraphia, or motor aphasia as a result. Occasionally the patient loses the memory of a certain period of time, and out of this a double personality may arise, the patient alternately recognizing one group of memories and then the other.

(*b*) *Aboulia* (absence of will power) is constant in hysteria. As a result the patient, is vacillating, impulsive, impressionable, and lacking in determination.

B. **Paroxysmal Phenomena.** 1. Convulsive attacks.

(*a*) *Hysteria major.* The severe, intense, and prolonged convulsive seizure (*grand mal hysterique*) described by Charcot is rare in this country. Such a paroxysm consists of a premonitory stage, in which various emotional and psychic disturbances may be present, followed by an aura (frequently the globus hystericus), and then an epileptoid seizure which closely mimics the true epileptic convulsion, and lasts about three minutes. At the close of this a period of clownism appears, the patient going through various contortions and wider-ranged movements. During this stage the patient

often utters violent outcries, grimacing, and biting, scratching, or striking at her attendants. The seizure may terminate in a period of passional attitudes, in which the patient depicts in pantomime love, rage, gaiety, or terror, or a period of delirium may supervene, the patient verbally expressing the hallucinations previously evidenced in pantomime. Finally, either suddenly or after a short period of silence, she arouses to her usual conscious state. The entire attack may consume from fifteen minutes to half an hour.

(*b*) *Hysteria minor.* Modified or partial seizures are frequently encountered. The more usual of these are attacks of vertigo, globus, epileptoid paroxysms, tetanic seizures, syncope, lethargy or trance, and somnambulism.

2. Motor Phenomena. Paralyses or contractures of a hysterical nature may follow convulsive seizures, shock, traumatism, or various morbid states. The paralyses often develop, either abruptly or gradually, after an emotional disturbance, but rarely is every movement of the extremity completely abolished. Antagonistic muscle groups are affected and the limb is perfectly limp; while the electrical reaction and reflexes are normal. Anesthesia or hyperesthesia may be associated. Hysterical contractures are characterized by loss of power and persistent involuntary rigidity, without change in the electrical or tendon responses. Among the most typical of hysterical motor phenomena are:

Hysterical hemiplegia, more frequent on the left side, and usually incomplete; often it affects only the distal portion of the limb. Instead of the rigidity of organic hemiplegia, the limb is flaccid; for example, in walking the leg is simply dragged along the ground.

Hysterical monoplegias may be single or multiple, unilateral or crossed. As a rule the member is overlaid by an area of anesthesia of greater extent than the paralysis.

Astasia-abasia is a hysterical condition in which coordination of the movements necessary for walking or standing is abolished. While in a bed or chair the patient has complete

control of her limbs, but is unable to stand, or, if standing, to walk. She may at the same time be able to run, leap, or hop. The condition is due to a form of amnesia, the motor image of walking being lost.

Hysterical *rythmical spasms* may be exemplified in periods of nodding, sneezing, grunting, or choreiform movements. Hysterical *tremors* may be of any variety, even the intentional form; and hysterical *tics*, movements of a purposive character, are sometimes seen.

3. Sensory Phenomena. The hysterical patient may suffer with persistent headache, spinal sensitiveness, visceral neuralgias, or even pseudo-angina pectoris. The resemblance of these disturbances to corresponding symptoms of organic disease is often close.

4. Visceral Phenomena. In hysteria the respiratory tract may be affected, stammering, coughing, dyspnea, and aphonia being sometimes induced. The last-named symptom is frequently due to a hysterical paralysis of the larynx.

The digestive system is usually involved, the symptoms varying from anorexia to esophageal spasm, and attacks of hysterical vomiting. Various abdominal disturbances may also be present, and in connection with contracture of the muscles a phantom tumor may be produced. Constipation is very common. Urinary disturbances, varying from anuria to the discharge, often after a hysterical crisis, of large quantities of limpid urine, may occur. Many hysterics are troubled with frequent urination. Even fever, may, though rarely, be of hysterical origin.

5. Trophic and Vasomotor Phenomena. These are few in number and quite rare; they consist of erythematous or vesicular eruptions, and local asphyxias similar to those of Raynaud's disease.

Diagnosis.—The diagnosis of hysteria must be based upon the complete exclusion of all possible organic causes for the phenomena present. The disease should be sharply distinguished from neurasthenia, simple emotional disturbances, and malin-

gering. The mental and psychic characteristics are often in themselves distinctive. In every case search should be made for the sensory stigmata; the disturbance of the color fields, and the peculiar outlines of anesthesia, with movability of the anesthetic areas, are practically pathognomonic. It must be remembered, however, that hysteria can coexist with organic disease, and in consequence such affections as tuberculosis, chronic nephritis, and cerebral hemorrhage must not be overlooked because of their development in a patient hitherto neurotic.

Prognosis.—Hysteria is essentially dependent upon a mental state, a temperament rather than a disease, and in consequence it cannot be wholly eradicated. Its paroxysmal phenomena are often controllable, however, particularly when influenced by certain mental and moral suggestions.

Treatment.—Since hysteria rests upon a mental basis, the treatment must be principally psychic. The physician must secure for himself and his measures the absolute confidence of the patient; and on this account the use of novel methods calculated to strike the fancy of the sufferer and inspire hopefulness is fully justified. In short, the treatment must be suggestive.

In order to accomplish this the patient should be isolated, and thus cut off from the friends and surroundings associated with the morbid past. The environment must be cheerful, and the attendants sensibly but not solicitously sympathetic, and at the same time optimistic. In severe cases these conditions are best secured by the rest cure in charge of a suitable nurse. In milder cases and in the young a prolonged journey with a sensible companion may meet all requirements. Medicines are of only secondary importance, but general conditions may afford indications for the administration of such remedies as *belladonna*, *cimicifuga*, *ignatia*, *nux vomica*, *pulsatilla*, and many others.

Special symptoms may be met by special measures. A convulsion may be checked by a sharp command, a dash of cold

water, or by pressure upon a hysterogenic zone. Paralyses and contractures, while often intractable, may be overcome if treated early by massage, electricity, and repeated suggestions of cure. Anesthesia and hyperesthesia may be abolished or modified by the use of faradism. Aphonia also may be overcome by the local application of faradism, coupled with manipulations and verbal suggestions of recovery. Dysphagia may be controlled by passing a stomach tube and thus proving that the esophagus is not obstructed.

EPILEPSY.

Etiology.—Epilepsy is in reality a generic term for a group of symptoms due to various pathological conditions. Among the more important predisposing causes are:

1. *Heredity.* A neurotic ancestry is an important factor in the production of epilepsy. Frequently the latter represents a transformation from other ancestral neuroses (hysteria, insanity) or from arthritis, syphilis, or alcoholism.

2. *Age.* It is uncommon for epilepsy to begin after the age of thirty, and a majority of the cases originate within the first twenty years of life. Epileptoid attacks occurring later in life should arouse a suspicion of syphilis, gross brain lesions, degenerative changes, or traumatism.

The exciting causes may be fright, toxic agents (especially alcohol), infectious diseases (exanthemata), head injuries, and reflex irritations (intestinal worms, pelvic diseases, nasal growths, eye strain, etc.).

Pathology.—While not yet clearly understood, epilepsy is unquestionably a disease of the cerebral cortex.

Symptoms.—Epilepsy may be characterized by convulsions more or less typical, by certain post-epileptic states, and by mental and physical degeneration.

1. *Epileptic convulsions.* The typical epileptic attack *(grand mal)* may be preceded by slight temperamental changes, mild motor or sensory disturbances, or other vague prodromes.

The attack is ushered in by an aura which may be motor (muscular contraction, tremor, automatic movement, etc.), sensory, a peculiar sensation arising in the abdomen or extremities and mounting to the head, or involving a special sense (visual or auditory sensations, etc.), or psychic (gaiety, rage, etc). This is immediately followed by a convulsion which presents three distinct periods: (1) A *tonic stage*, the patient uttering a cry and falling in a state of tetanic rigidity which lasts but a minute or two, and is followed by: (2) A *clonic stage*, in which the muscles alternately relax and contract, thus causing a horrible twitching which usually involves the entire body. This continues for from one to five minutes, when it is succeeded by: (3) A *stage of stertor*, in which the convulsive movements cease and the patient lies comatose and breathes stertorously. After a few minutes, perhaps half an hour or more, he gradually regains consciousness but has no knowledge of what has happened. During the convulsive period, the tongue may be bitten and the contents of the bowel and rectum expelled.

Modifications of this typical fit are not uncommon; the clonic movements may be confined to one extremity, or to one side of the body, or the cry, stertor, or urination may be lacking. In some cases the attacks occur only during sleep *(nocturnal epilepsy)*, and thus the disease may exist unsuspected for years. Occasionally the patient has a series of fits one after the other without recovering consciousness *(status epilepticus)*.

2. *Incomplete attacks (petit mal)*. Almost any part of the attack may occur alone; for instance, the patient may be seized only with an aura, or with vertigo, or with a momentary loss of consciousness. In rare instances the patient is seized with an irresistible impulse to walk or run, and this latter phenomenon sometimes immediately precedes or follows a typical convulsion. Certain psychic phenomena, automatic actions of a homicidal or obscene character, may appear in place of or in alternation with the typical attack (*psychical epilepsy*). The physical condition of the average

epileptic is good, except for the gastro-intestinal and other disturbances induced by the bromides or similar drugs with which he has been dosed; but his mental state undergoes progressive deterioration.

Diagnosis.—As a rule the typical attacks offer no diagnostic problem, but incomplete seizures may be extremely difficult of recognition. Suspicion should be aroused by a history of bed-wetting, unexplained bruises or ecchymoses, a bitten tongue, etc., and in such a case attacks of momentary unconsciousness or of sensory aura are extremely significant. Repeated convulsions of epileptic type are rarely mistaken for anything else. *Organic brain disease* may produce epileptoid convulsions, but such cases are distinguished by a history of headache, pain, vomiting, vertigo, optic neuritis, and other symptoms of cerebral disease. As a rule, the patient is older, the attacks are more protracted than those of epilepsy, consciousness may not be entirely lost, and sometimes the convulsion is Jacksonian in type and is followed by paralysis of the previously convulsed parts. *Uremic convulsions* closely resemble those of epilepsy, but as a rule they occur in later life, the seizures are prolonged, and the urine is apt to contain albumin, casts, and a deficient amount of urea.

For the differentiation of hysterical and epileptic seizures, the following table (Church) may be consulted:

	Epilepsy.	*Hysteria.*
Prodromes.	Mental or physical premonitions.	Emotional disturbance.
Aura.	Common but momentary.	Uncommon and of considerable duration.
Onset.	Sudden, complete; cry, fall, rigidity.	Gradual.
Consciousness.	Instantly lost.	Partially lost or retained.
Course.	Tonic, clonic, and stertorous stages.	Epileptoid and emotional phases.
Duration.	Two to five minutes.	A few minutes to several hours.

	Epilepsy	*Hysteria.*
Positions.	Governed by flexors mainly.	Tendency to extension, *arc de circle*, opisthotonos, crucifixion attitude, etc.
Eyes.	Pupils dilated and rigid.	Pupils mobile and active.
Tongue.	Usually bitten.	Bitten very exceptionally.
Mouth.	Frothing common.	Absent.
Sphincters.	Relaxed usually.	Usually continent.
Pulse	Accelerated greatly and tension increased.	Rate and tension not much changed.
Temperature.	Elevated 1° or 2° F.	Normal.
Post-paroxysmal conditions.	Petechial ecchymoses, reduced muscular strength, diminished knee-jerks, mental obscuration, no memory of attack.	No motor or reflex changes; some recollection of phases of attack; usual mental condition at once regained; presence of various stigmata.

Prognosis.—Recovery is rare, and after the disease has become thoroughly established there is scarcely any hope of cure. Death may occur in a status epilepticus, or as a result of injury received during a convulsion. Otherwise, the natural termination of the disease is in dementia.

Treatment.—Study the patient thoroughly. Remove every possible source of reflex irritation, correcting eye strain, nasal disease, and dental, gastro-intestinal, and genital troubles. Regulate the diet so that it is nourishing and digestible; allow vegetables, fresh fruit, milk, and fish, a limited amount of starchy food, and little meat. Direct the patient to bathe daily, to live a vigorous out-door life as far as possible, and in other respects regulate his hygiene. The more important medicines are *argentum nit.*, *belladonna*, *bryonia*, *calcarea carb.* *cicuta*, *cuprum*, *hydrocyanic acid*, *hyoscyamus*, *nux vomica*, *œnanthe*, *plumbum*, *pulsatilla*, and *sulphur*. Only as a last resort, these having failed, is it justifiable to resort to the bromides of potassium, sodium, or strontium; of these, from fifteen to thirty grains may be given daily in divided doses, each of the latter being dissolved in a large quantity of water aud taken after meals.

NEURASTHENIA.

(Nervous Prostration; Nervous Exhaustion).

Etiology.—Neurasthenia is a condition of "irritable weakness," a neurosis in which all forms of nervous energy—psychic, motor, and organic—are reduced, as a result of fatigue of the nerve cells. The more important predisposing causes are:

1. *Age.* The condition seldom develops before the twentieth or after the fiftieth year of life; it is a disease of the energetic period.

2. *Heredity.* While neuropathic antecedents are less common than in hysteria or epilepsy, nevertheless debilities in the parents are apt to weaken the offspring and so lessen the endurance of the latter.

The exciting cause of neurasthenia is usually overwork, often associated with worry and excitement, alcoholic or sexual excesses, exhausting illness, and such toxic states as gout or syphilis. Shock, either traumatic or psychic, may be a direct cause.

Symptoms.—The general depression of nervous energy gives rise to a host of symptoms, the more important of which may be classified under the following heads, viz.:

1. *Motor disorders.*—Muscular weakness is a constant complaint, the patient becoming fatigued on the slightest exertion. Tremors and muscular twitching are common, and the tendon reflexes are increased.

2. *Sensory disturbances.*—The patient feels generally tired and fatigued. He complains of headache, often at the base of the brain, and at other times of a terrible weight, like a lead cap, over the head. Backache is equally common. Tenderness over the spine is usually present, but cutaneous anesthesia, never. Many vague subjective sensations, such as numbness, prickling, soreness, etc, are encountered.

3. *Gastro-intestinal disturbances.*—Nervous dyspepsia, with the various symptoms and signs peculiar to that disorder, is characteristic of neurasthenia. Constipation, due to atony of the intestines, is also common.

4. *Circulatory disorders.*—These patients frequently suffer with palpitation, and even with attacks of pseudo-angina. Often the circulation is feeble, the pulse rapid and the extremities cold, and vasomotor symptoms, such as flushes and sweats, occur. The fluid secretions may be scanty. The hand is usually cold and clammy with perspiration.

5. *Disorders of special sense.* Reading causes headache and distress, the accommodative apparatus and the retinal sensibility becoming quickly fatigued. Photophobia and hypersensitiveness to sounds, smells, and tastes are common symptoms.

6. *Genital disorders.* The male neurasthenic usually complains of lessened sexual power, or he may be frightened by nocturnal emissions. If he has at any time masturbated, his time is spent brooding over that and its supposed effects, especially his impotency (*sexual neurasthenia*).

7. *Mental disturbances.* The capacity for mental work is reduced, the memory is poor, and as a result the patient becomes introspective and hypochondriacal. Morbid fears and emotional outbreaks are common, while insomnia is almost habitually present.

Diagnosis.—The diagnosis of neurasthenia is generally easy, but it must be remembered that the condition is frequently merely symptomatic of some organic process, *e. g.*, following any exhausting illness or accompanying anemic or cachectic states. It is frequently associated with *hysteria*, and so closely may the two resemble each other that it is only by discovery of the hysterical stigmata that the distinction can be made. *Hypochondriasis* is in reality a melancholia, the patient suffering with delusions concerning his bodily state.

Prognosis.—Neurasthenia is essentially a chronic disease, insidious in its onset and tending, if untreated, to persist indefi-

nitely. If proper treatment, *i. e.*, protracted rest and change, can be secured, the outlook is usually favorably. Relapses are common, however.

Treatment.—1. *Rest.* This constitutes the one essential factor for successful treatment. According to the severity of the malady and the temperament of the patient, this rest may be partial, the patient retiring immediately after the evening meal and sleeping late in the morning, or complete, the patient being placed at absolute rest under the care of a nurse for a period of several weeks. In every case the amount of work must be reduced, and frequently this is best accomplished by sending the patient on a vacation which entails prolonged separation from business and provides a maximum of fresh air and recreation without undue physical exertion. For this purpose a sea voyage is admirable.

2. *Diet.* A nourishing, non-stimulating diet which tends to fatten the patient should be insisted upon. It matters not what he eats, provided enough is taken and digested. A glass of hot milk or beer at bed time encourages sleep.

3. *Hydrotherapy.* A warm bath of five or ten minutes' duration should be taken immediately before retiring. A cold spinal douche or a quick cold sponging on arising is invigorating.

4. *Massage* is essential when the complete rest treatment is carried out; in other cases it is not so necessary, but unquestionably aids the nutritional progress.

5. *Environment.* On account of the mental element present in neurasthenia, the patient's surroundings should be cheerful and his attendants should offer constant hope and encouragement. The physician should see him frequently, explaining away his fears, but refusing tactfully to let him reiterate his complaints.

6. *Medicines.*—Among the many useful remedies are *argentum nit.*, *cimicifuga*, *nux vomica*, *phosphorus*, *phosphoric acid; picric acid*, *platina*, *sepia*, *strychnine*, *sulphur* and *zinc.*

CHOREA.

(Chorea Minor; Sydenham's Chorea.)

Etiology.—Chorea must be carefully distinguished from other choreiform affections, such as habit chorea, hereditary chorea, the various forms of tic and myoclonus, and the hysterical manifestation properly known as St. Vitus's dance. The more important predisposing factors are:

1. *Sex.* Girls are affected much more frequently than boys.

2. *Age.* The great majority of cases develop between the fifth and fifteenth years of life.

3. *Heredity.* The neurotic heredity plays an important part in the production of this, as of the other neuroses.

4. *Season.* Chorea is most frequent in the winter and spring months, thus corresponding to rheumatism and other diseases whose prevalence is affected by climatic vicissitudes.

Among the exciting causes have been listed fright, worry, shock, overwork, and reflex irritation from the intestines. This relationship is in all probability simply coincidental, for the trend of modern investigation is to demonstrate that chorea, which is so commonly associated with more or less typical rheumatic manifestations, is due to a similar infective agent.

Pathology.—The disease is probably localized in the cerebral cortex, although no distinctive lesions have been demonstrated. In fatal cases the heart lesions characteristic of rheumatism (endocarditis, pericarditis) are usually discovered,

Symptoms.—The disease develops insidiously, the child first becoming peevish, inattentive, and depressed. This is followed after a varying period by—

1. *Choreic movements.* These are sudden, involuntary, slight movements which make the child appear maladroit, upsetting his cup, dropping things from his hand, etc. Twitching of the facial muscles causes various grimaces, and the involvement of the muscles of the tongue may render speech indistinct and swallowing difficult. Station may be unsteady

and the gait irregular because of the twitching of the muscles of the lower limbs. Involvement of the thoracic muscles and diaphragm may cause irregular breathing and sometimes spasmodic noises. Muscular power is always decreased, and in many cases this amounts to an actual muscular paresis.

2. *Mental disturbances.* Mental activity is lessened, the patient is forgetful, and often the face appears inane and stupid. Visual hallucinations may be present, particularly at night; and in the grave form elevation of temperature may be accompanied by delirium.

3. *Cardiac disorders.* Cardiac murmurs, both functional and organic, are extremely common in choreic patients. Endocarditis, sometimes associated with joint symptoms, occurs in more than half of the cases; and the resulting valvular lesions, of which mitral insufficiency is the more frequent, persist throughout life.

Forms.—In addition to the ordinary variety just described, three others may be distinguished, viz.:

The *grave form* (*chorea gravis*) is acute in its onset, marked by severe mental and motor phenomena, and accompanied by fever and delirium (*choreic status*). This persists for days and frequently terminates in coma and death.

The *chorea of pregnancy* (*chorea gravidarum*) appears especially in young primiparæ, presents intense motor agitation, and usually persists until the uterus is emptied. It is often fatal (20%), or it may recur with subsequent pregnancies.

Paralytic chorea is characterized by the predominance of muscular paresis. It may be monoplegic, hemiplegic, or paraplegic in its distribution. The muscles are toneless, and the reflexes are abolished, but the reaction of degeneration is absent, and recovery within a few weeks is the rule.

Diagnosis.—The symptoms usually facilitate a diagnosis, but it is necessary to distinguish chorea from the other diseases presenting choreiform movements. *Athetosis*, *post-hemiplegic chorea*, and *Friedreich's ataxia* may be recognized by their cerebral associations. *Huntingdon's chorea* has a history of

heredity, appears in adults, and is accompanied by progessive dementia. The various *tics* are more sudden and are gesticulatory in character.

Prognosis.—As a rule, chorea gradually declines, and in the course of from one to four months terminates in recovery. Recurrences are very common, however, and the possibility of serious cardiac complications must also be remembered. The grave form is usually fatal, and the chorea of pregnancy often proves fatal to the mother, and yet more frequently destroys the fetus.

Treatment.—1. *Rest.* As a rule the child should be kept at home and away from violent romping or excitement. The hours of sleep should be prolonged, and frequent sponge baths and warm general baths and gentle massage are helpful. The more important medicines are *agaricin*, *actea*, *belladonna*, *causticum*, *cuprum*, *ferrum*, *hyoscyamus*, *iodine*, *mygale*, *nux vomica*, *pulsatilla*, *stramonium*, *tarantula*, and *zinc*.

The severe cases require absolute rest in bed, forced feeding is often advisable, and hot baths or cold packs should be used to meet the same indications as in any infectious fever. In extreme cases it may be necessary to resort to chloral, bromides, or even morphia. In the chorea of pregnancy, violent motor manifestations sometimes, though rarely, render render it necessary to terminate gestation.

TETANY.

(Tetanella.)

Etiology.—This peculiar tonic spasm of bilateral groups of muscles is rare in America, but quite common in continental cities. It appears usually before the twentieth, always before the fiftieth year of life. The principal predisposing factor is impaired nutrition, due to gastro-intestinal diseases, infections, pregnancy, myxedema, etc. The attack may be precipitated by exposure, exhaustion, or emotional disturbance, although the existence of an infective organism is suspected.

Symptoms.—The attacks are initiated by a sensation of numbness and prickling in the fingers or toes, followed by a tonic spasm affecting chiefly the flexor muscles of both arms and both legs. The attacks may last for minutes or hours, seldom days. Several paroxysms usually occur daily. Intervals of days, weeks, or months may separate the seizures, but the latter can be provoked at any time by pressure over the larger nerve trunks and arteries of the arm (*Trousseau's phenomenon*). A facial spasm, limited to the distribution of the nerve or branch pressed upon, can be aroused in similar fashion (*Chvostek's sign*). Electric irritability, especially to the galvanic current, is exalted (*Erb's phenomenon*). The temperature is usually normal, and cutaneous sensibility and the reflexes are unaffected.

Diagnosis.—The characteristic spasms, accompanied by mechanical and electrical over-excitability of the nerves, render a mistake improbable. *Tetanus* begins with masticatory stiffness, which does not subside in the intervals between spasms; the disease begins in the trunk, frequently sparing the hands; and the peculiar phenomena of tetany are absent. *Hysteria* also lacks the latter signs and presents its own peculiar stigmata.

Prognosis.—The disease usually runs a mild course, recovery ensuing in a few weeks; but in some cases in which the cause of the underlying malnutrition is not removable (*e. g.*, dilatation of the stomach) it may be protracted or even produce death by asphyxia. In connection with myxedema strumipriva it is very fatal (80%). In other cases it tends to recur.

Treatment.—The treatment should be directed to the cause of the underlying malnutrition. Quiet, rest and warm baths aid in controlling the attacks. *Belladonna*, *magnesia phos.*, *nux vomica*, and *secale* have been recommended, but in general remedies should be prescribed for the causative condition.

CHOREIFORM DISEASES.

Huntingdon's (Hereditary) Chorea appears in adult life, between the ages of thirty and fifty, as a result of direct heredity. The

twitchings begin gradually in the upper half of the body, increasing in distribution and intensity. The disease is accompanied by a progressive mental impairment which terminates in dementia. The affection is chronic and incurable, and its pathology is unknown.

Habit Chorea (**Habit spasm**) is a condition presenting definite spasmodic movements, such as blinking the eyes, twitching the nose or the corners of the mouth, or shrugging the shoulders. In children the prospect of recovery from the habit is good, but should the disease persist into adult life it is not so promising.

Electrical Chorea (*Dubini's disease*) is a very rare affection which occurs in certain districts in Italy. It is characterized by sharp spasmodic muscular movements, similar to those produced by electrical discharges, which involve each of the extremities in succession. The muscles become weak and undergo atrophy, general paralysis ensues, and the patient dies.

Myoclonus (*Paramyoclonus multiplex*) is a term comprehending a group of diseases characterized by involuntary, sharp contractions of a muscle or group of muscles, similar to those produced by an electric shock. They are usually bilateral in character and arhythmic. They involve first the lower limbs then the upper, but are apt to spare the face. The course extends over months, but recovery generally occurs. *Tics* are to be distinguished; they are usually at first unilateral, affect the face, and are purposive, expressive, or gesticulatory in character.

PARALYSIS AGITANS.

(Parkinson's Disease; Shaking Palsy.)

Etiology.—The factors predisposing to paralysis agitans are age (40–70 years), sex (two-thirds of the patients are males), and a neuropathic heredity. Fear, anxiety, grief, exhaustion, shock and traumatism have been named as exciting causes.

Pathology.—While the disease is classed as a neurosis, it is probable that vascular changes in the peduncular circulation, leading to degenerative processes, underlie the disease.

Symptoms.—The disease begins with rigidity in one arm and tremor of the fingers; and these symptoms extend first to the foot of the same side, then to the hand and foot of the opposite side. Ultimately there is stiffness of the whole body. The symptoms in a typical case are very striking; they include:

1. *Muscular rigidity.* The attitude of the patient is characteristic: his head and body are bent forward, his neck is extended, and he walks or trots with short, shuffling steps as though following his centre of gravity (*propulsion*). His elbows are flexed and slightly abducted, bringing his hands to the level of the groins. The rigidity may impede passive motion, but the reflexes are only slightly increased and clonus never develops. The stiffness of the muscles renders the face immobile and mask-like; and speech is monotonous and deliberate.

2. *Tremor.* The tremor may appear with or after the rigidity. The patient appears to be rolling a pill between his fingers and thumb. The tremor affects all the extremities, but seldom involves the head muscles. It continues when the member is at rest, but ceases upon voluntary motion, and during sleep. The tremor makes itself apparent in the handwriting.

Diagnosis.—Typical cases are diagnosed at a glance. In *senile tremor* the head is first affected, rigidity is lacking, and the patient is advanced in years. *Disseminated sclerosis* presents an "intention" tremor, increased reflexes, scanning speech, and nystagmus. A *post-hemiplegic tremor* has a history of a stroke, together with unilateral paralysis and increased reflexes.

Prognosis.—The prognosis for cure is unfavorable, but the patient may live for many years.

Treatment.—Treatment should be directed to the cerebral arterio-sclerosis in order to check the progress of the lesion, and in the meantime symptomatic prescriptions of such remedies as *agaricin*, *mercurius*, *plumbum*, *phosphorus*, and *tarentula* are advisable. In severe cases the tremor may some-

times be controlled by the administration of *hyoscin hydrobromate* 2x or *hyoscyamine* 2x, in one grain doses three times a day.

OCCUPATION NEUROSES.

(Fatigue Neuroses; Writer's Cramp, etc.).

Etiology.—An occupation which requires the constant repetition of certain precise muscular movements may eventually lead to fatigue of the sub-cortical motor centre, and this results in disturbance of the muscular control necessary for that particular maneuver. Writers, pianists, telegraphers, seamstresses, and many others are liable to this neurosis.

Symptoms.—The onset is usually insidious. The worker notices that after a time his hand aches or feels numb, or cramped, or tremulous. After a moment's rest he resumes his work, but the trouble recurs; and less and less work becomes necessary to induce the disablement, until finally the condition is permanent and he is completely incapacitated. The power for all other movements remains unimpaired. If no attempt is made to resume the occupation the cramp ultimately disappears; but usually it returns upon any endeavor to take up continuously his former work.

Diagnosis.—The history, and the provocation of the symptoms only by attempts at resuming the occupation, render the diagnosis clear.

Prognosis.—If the patient can completely abstain from his customary occupation, the spasm tends to subside and disappear. He cannot hope to ever again be able to resume his former method of work, however.

Treatment.—The affected muscles must be put at rest, and various exercises, massage, electricity, and baths may be employed for their general tonic effect. If his vocation cannot be changed, the sufferer should learn to use the opposite hand, or else some specially constructed apparatus by which the strain is taken from the affected motor area. Among the more useful remedies are *arnica*, *gelsemium*, *phosphorus*, *rhus tox.*, and *strychnine*.

MIGRAINE.

(Hemicrania; Sick Headache.)

Etiology.—This paroxysmal psychoneurosis is obscure in nature, though probably cortical in origin. A neuropathic heredity and the gouty diathesis seem to act as predisposing causes, and it usually makes its appearance in early life, between the ages of five and ten years. The exciting cause is often difficult to determine; sometimes it is eye strain, and in other cases depressed nutrition, indigestion, constipation, fatigue, emotional disturbances, lithemia, etc.

Symptoms.—The only symptoms of migraine are those of the attack itself. While these vary somewhat, the following stages usually present themselves:

1. *Premonitory symptoms.* The patient may for some hours be dull and irritable, or suffer with a slight headache.

2. *Sensory symptoms.* Suddenly bright spots, colored rings, and zigzags or clouds appear before the eyes, or the attack may be ushered in by simple dimness of vision. Exceptionally taste or hearing is affected, or numbness and tingling are felt elsewhere in the body. Rarely motor symptoms, such as paresis or motor aphasia, occur at this stage.

3. *Headache.* In the course of ten or twenty minutes the headache begins. It is usually intense and at first localized to one spot, whence it spreads over the rest of the same side or involves both sides of the head. It is accompanied by marked hyperesthesia of all the senses, and may last for one or many hours.

4. *Nausea and vomiting.* As the headache reaches its height, nausea generally develops and in many cases is followed by vomiting. The latter may be severe and protracted, but as it subsides the headache is apt to lessen and the final stage, that of

5. *Sleep*, supervenes. After a few moments or perhaps several hours of slumber the patient awakens free from pain.

Vasomotor symptoms, such as flushing, pallor, and perspiration, sometimes accompany the paroxysm.

Diagnosis.—The periodical character of the attacks, the sensory symptoms with which they are introduced, and the nausea and vomiting, serve to distinguish the disease. Even in cases less typical the occurrence of these sensory symptoms is sufficient to identify the disease. Occasionally it may be necessary to exclude organic brain lesions, uremia, or malaria; but this is rarely difficult.

Prognosis.—The prognosis is generally unfavorable. If the disease is of recent onset, and the cause can be discovered and removed, recovery may ensue. Otherwise it is apt to persist in women until the menopause, and until a corresponding age in men, when it disappears spontaneously.

Treatment.—Search for, and if possible remove, the cause. Correct eye-strain, improper diet or habits, and all other probable sources of irritation. The more important internal remedies, the administration of which should be continued in the interval between attacks, are *belladonna*, *calcarea*, *cannabis indica*, *gelsemium*, *ignatia*, *iris*, *nux vomica*, *sanguinaria*, *sepia*, *stannum*, and *sulphur*. One drop of *glonoin* 2x taken on the tongue will sometimes abort an attack, and in others *caffein* gr.j-v, has a similar effect. During the paroxysm the patient should remain at rest in a darkened, quiet room.

TRAUMATIC NEUROSES.

Varieties.—Three classes of conditions may be due to traumatism, viz.:

1. Injuries of a surgical character.
2. Traumatic hysteria.
3. Traumatic neurasthenia.

Various combinations of these three varieties may also present themselves.

Diagnosis.—Since these conditions are frequently the subject of litigation, it is essential that malingering be carefully ex-

cluded. For this purpose it is necessary, if no evidence of an actual surgical lesion is present, to apply to the patient the same diagnostic methods that would be used for the investigation of cases of hysteria and neurasthenia due to other causes. The **prognosis,** when complicated by the question of obtaining damages for the injuries received, is less favorable than in simple cases; so that prompt settlement of the dispute is desirable from a physician's standpoint. The **treatment** is that of hysteria and neurasthenia whatever their causes.

TICS.

(Mimic Spasm).

Etiology.—A tic is a spasm identical with a volitional movement, and probably of subconscious origin. It is apt to appear in youth, but no age is exempt; and it frequently represents the habit induced by some local irritation (*e. g.*, sniffing due to a nasal catarrh, blepharospasm induced by ocular irritation). Protracted emotions may induce similar tendencies to involuntary facial contortion.

Symptoms.—These vary according to the location of tic; the more important of the latter are as follows:

Facial tic. This consists of an involuntary lightning-like spasm of a muscle or set of muscles, such as blinking the eye (*blepharospasm*), twitching of the nose, grunting, or even uttering articulate words (*coprolalia*). The facial expression of joy, grief, fright, or pain may thus be produced, often as an indication of some subconscious idea. This spasm may involve the neck and upper extremities, occasioning various attitudes and gestures.

Spasmodic torticollis (*wry neck*). This is a spasm of the muscles innervated by the spinal accessory and sometimes those supplied by the upper cervical nerve, as a result of which the neck is drawn to one side. It is distinct from wry neck due to injury or atrophy of the sterno-mastoid before birth (*congenital torticollis*), from wry neck due to rheumatic myositis, adenitis, abscess, etc. (*symptomatic torticollis*), and

from spasm of the neck muscles due to spinal caries (*spurious torticollis*). *Mental torticollis* is marked by a customary deviation of the head which is spasmodically maintained, but which disappears when the patient lies down, or it can be controlled by slight manual pressure.

Jumpers and subjects of *latah* and *myriachit* impulsively obey certain suggestions, often violently and apparently against their will, *e. g.*, striking or jumping.

Treatment.—Correct any possible cause for peripheral irritation. Pressure upon certain points in the distribution of the fifth nerve inhibits a facial tic; search for such points, and having found them, repeatedly check the tic in order that ultimately the habit may be arrested. Faradism may have a good effect, and even nerve stretching may be employed. The patient's self-control should be built up and reinforced by suggestion.

TROPHONEUROSES.

ACROMEGALY.

Etiology.—The cause of this disease is practically unknown. It usually develops between the ages of eighteen and thirty, and is frequently associated with disease of the pituitary body.

Pathology.—The bones of the face, cranium, and extremities, and to a certain extent those of the trunk, become hypertrophied. The skin and mucous membranes are thickened with connective tissue, the kidneys, spleen, and lymphatic glands may be sclerosed, the thyroid is often atrophied, and the thymus may be persistent and even increased in size. The pituitary gland is enlarged and hypertrophic.

Symptoms.—The head is greatly enlarged and the face is strikingly deformed, the thickened skin with deepened furrows, the thick lips, and the coarse hair giving the features a heavy, dull expression. The hands and feet are markedly enlarged, especially in width. The complexion is usually sallow, and the skin is doughy and does not pit on pressure. The

patient is apt to suffer with intense persistent headache; vision is affected, speech may be thick, and sexual power is lost. As a rule he is physically weak and mentally sluggish. Polyuria, glycosuria, and dyspepsia are common. The reflexes are normal or diminished and the electrical reactions may be quantitatively reduced.

Diagnosis.—The disease is slow in its development, extending over a period of years, and in its earlier stages may be difficult of detection; but when the physical changes are marked an error is improbable. *Myxedema* exhibits a thickened skin but no bony deformities.

Prognosis.—The disease, after a duration of twenty or thirty years, terminates in muscular weakness and cachexia, and sudden death from heart failure is frequent.

Treatment is of no avail. At best it can only relieve symptoms, such as headache, by means of appropriate medicines.

RAYNAUD'S DISEASE.

(Symmetrical Gangrene).

Etiology.—The causes of this vasomotor disorder are very obscure; inmany cases anemic and neurotic states appear to predispose. The attack may be excited by exposure, severe emotional disturbance, or acute infectious diseases.

Symptoms.—The most striking symptom is the pale, bloodless, waxy condition (*local syncope*) of the fingers and toes, less often the ears, nose, or lips. A needle-prick draws no blood, and the fingers appear dead (*digiti mortui*). There is usually a sensation of numbness and tingling, with loss of sensibility. In a few minutes or hours this may pass off, but in many cases a second stage develops, the affected part becoming cyanotic and blue-black or red (*local asphyxia*). In a few hours small blebs may appear, and are often followed by ulceration or by a dry gangrene which destroys the terminal phalanges or even entire fingers. This stage is accompanied by neuralgic pain.

Constitutional symptoms, such as intermittent hemoglo-

binuria, colic, or uremic convulsions or coma, are frequently present.

Diagnosis.—Care should be taken to exclude gangrene due to other causes, including senile gangrene, frost-bite, ergot-poisoning, and obliterating endarteritis.

Treatment.—Treatment must be directed toward improvement of the general health. During the attack, heat or electricity may be used locally, and *glonoin* given internally to relieve the arterial spasm. *Arsenic* or *secale* may also be indicated.

ANGIONEUROTIC EDEMA.

Angioneurotic edema (acute circumscribed edema, giant urticaria) is a disease of unknown causation characterized by the sudden appearance upon the face, arms, or hands of a circumscribed swelling, pale or red in color, and pitting only slightly on pressure. There is no actual pain, but itching, burning, or stiffness is noted. The swelling disappears in a few hours or days, but may recur frequently. By involving the larynx (*edema glottidis*) it may prove fatal; otherwise, the prognosis for life is good, but for cure doubtful. The **treatment** must be directed to the patient's general condition, and especially to any disorder of the digestive system.

SCLERODERMA.

Scleroderma is a rare disease characterized by marked induration of the skin. The condition is due to the proliferation of connective tissue in patches, or in widely diffused areas. The thickness of the skin, together with a certain degree of muscular atrophy induced by the pressure, results in stiffness and impaired mobility. The course of the disease is very chronic. The **treatment** consists of massage, oil inunctions, galvanism, warm clothing, and the administration of *antimonium crud.*, *bryonia*, *guaiacum*, *graphites*, *hydrocotyle*, *lachesis*, *phosphorus*, *silica*, or *stillingia*.

FACIAL HEMIATROPHY.

Facial hemiatrophy is a rare disease characterized by gradual atrophy of one-half of the face, the wasting stopping short at the median line. The muscles are not paralyzed and the electrical responses are unaltered. The disease must be distinguished from atrophic palsies due to lesions of the fifth nerve, in which paralysis and the reaction of degeneration are present. The cause of the disease is unknown. It progresses slowly over a long period of time, but finally becomes stationary and does not endanger life. **Treatment** is unavailing.

INFANTILE CONVULSIONS.

(Infantile Eclampsia.)

Etiology.—Owing to lack of development of the higher cerebral centres in children, the inhibition exercised by them upon the lower centres (in the pons Varolii) is weak, and the latter react to comparatively slight irritations. In consequence, convulsions are common in early childhood, especially during the first and second years of life, but rapidly decrease in frequency as age advances. A neuropathic heredity strongly predisposes its victims to convulsive seizures in infancy.

An infantile convulsion is only a symptom. It may be due to:

1. *Gastro-intestinal irritation.* This may be the result of dietetic errors, indigestion, gastro-enteritis, or the presence of worms.

2. *Peripheral irritation.* Teething, otitis, phimosis, a slight burn, or a mere prick of the skin may cause a convulsion. A foreign body in the respiratory passages, digestive tract, or ear may have the same effect.

3. *Acute febrile diseases.* The onset of an acute infection, notably pneumonia or the exanthemata, may be accompanied in children by convulsions.

4. *Disease of the brain or its membranes.* Circulatory dis-

disturbances, such as the mechanical congestion following violent coughing, meningitis, and gross lesions of the brain, especially those inducing the infantile cerebral palsies, may be the cause of convulsions.

5. *Rickets* is frequently accompanied by recurrent convulsive seizures.

6. *Epilepsy* is not seldom the cause of infantile convulsions.

Symptoms.—As a rule the onset is abrupt, the child becoming pale and rigid. Its eyes are fixed, and the eye-balls are rolled upward so that only the whites are visible, or there may be strabismus. The fingers and toes are inverted, and the face becomes cyanotic as the result of impaired respiratory movement. The child loses consciousness, and it may have involuntary evacuations. The spasm is usually tonic, but it may become clonic, as in epilepsy, or it may be so from the beginning. Generally at the end of two or three minutes the muscles relax, and the child falls into slumber.

Diagnosis.—The symptoms are unmistakable, but the question of etiology is sometimes difficult of solution. Convulsions due to indigestion may be limited to a single attack or series of attacks. If there be much fever, the onset of an acute infectious disease may be suspected. If the convulsions are due to a brain lesion, they are apt to be repeated and to alternate with paralysis. Attacks occurring irregularly and without definite cause in children over two years of age are probably epileptic.

Prognosis.—As a rule, in cases due to simple irritation the prognosis is favorable. In weakly patients, however, death may be brought about by exhaustion. In convulsions symptomatic of other diseases the prognosis is governed largely by the character of latter.

Treatment.—At the moment of the attack loosen the clothing, empty the bowel by means of an enema of soap and water or oil, and immerse the child in a warm bath (95°). If the attack is long-continued, apply cold to the head. Search for the cause and remove it if possible. The convulsion itself may present indications for the administration of *belladonna*,

cuprum, *cicuta*, *ignatia*, or *magnesia phos*. If it is but a symptom of another disease, however, the treatment should be directed to the latter.

DISORDERS OF SLEEP.

Insomnia is a condition of sleeplessness which may accompany almost any deviation from health. In certain cases it becomes a fixed habit, and may be the only complaint of the patient. Ultimately the lack of proper rest leads to loss of energy, courage, good nature, appetite, and digestion; and as a result the patient becomes haggard and emaciated. **Treatment** must first be directed to the removal of the causative condition (drugs, narcotics, lithemia, uremia, infectious diseases, neurasthenia, intense mental activity, painful affections, etc.), if it can be discovered. Improper methods of living should be corrected, and appropriate food should be eaten. Sleep may be favored by warm baths, not followed by stimulating friction, at bed-time. Occasionally a cold pack or an alcohol rub acts well. A glass of hot milk, malted milk, or beer at bed-time is usually advisable. The administration of *belladonna*, *cannabis indica*, *coffea*, *hyoscyamus*, *opium*, *pulsatilla*, or *stramonium*, when symptomatically indicated, is usually all the medication advisable. Remedies powerful enough to induce sleep are necessarily dangerous and must not be used with impunity. In exceptional cases, however, the physician is justified in prescribing *chloral hydrate*, gr. x-xv, and if there is much nervousness, an equal quantity of *sodium bromide*, in a single dose at bed-time. *Sulphonal*, gr. x-xx dissolved in a glassful of hot water, or a similar quantity of *trional*, may be preferred in some cases.

Somnambulism is a condition in which the individual, in enacting his part in a dream, arises and moves about while asleep. He retains no waking knowledge of this sleep-walking, but the memory of it may be revived during subsequent attacks. The condition usually appears in persons of neurotic habit, and treatment should be directed to the patient's general state rather than to the single symptom.

Dreams are perfectly physiological up to a certain point, but in connection with lowered physical or mental states they may tend to become depressing, terrifying, and distinctly pathological. In children *pavor nocturnus (night-terrors)* is not uncommon. The little one awakes from sleep screaming with terror and clings to its mother, and some moments elapse before it recognizes its surroundings and can be reassured. A similar, though milder, attack is the *nightmare (incubus)* of adults, in which a vivid, terrifying dream disturbs the sleep. In many cases these attacks can be traced to indigestion, imperfect ventilation, worry, fright, etc. Pavor nocturnus is, however, an indication of serious neurotic instability, and in a few cases it appears to be an early manifestation of epilepsy. **Treatment** in these cases should, in general, be directed to the discovery and removal of causative factors. The meals should consist of proper food at appropriate intervals, the condition of the alimentary tract should receive attention, and indications will be found for the administration of such medicines as *aconite*, *belladonna*, *cina*, *hyoscyamus*, *nux vomica*, *pulsatilla*, *spigelia*, *stramonium*, *sulphur*, and *zinc*.

Narcolepsy, a condition in which the patient repeatedly falls asleep during the day, may be a manifestation of hysteria, or it may be due to drugs, obesity, uremia, diabetes, syphilis, or organic brain disease. **Treatment** must be directed to the cause.

Catalepsy, ecstacy, and **trance** simply represent a prolongation of certain stages of the hysterical convulsion.

SUNSTROKE.

As the result of exposure to excessive heat two conditions may occur, viz. :

HEAT EXHAUSTION.

(Heat Prostration.)

Symptoms.—Heat exhaustion may result from exposure to intense heat in confined places, such as boiler-rooms or kitchens, as well as to the direct rays of the sun. The patient becomes

faint, dizzy, nauseated, and sometimes blinded; the skin is pale, cold, and covered with clammy perspiration, the pulse is rapid and feeble, and the temperature is subnormal. The patient may quickly become delirious or comatose, and actual collapse may ensue. The symptoms are supposed to be due to vasomotor paresis, as a result of which the blood collects in the great abdominal vessels and leaves the brain and the body-surface bloodless. In the absence of medical intervention, a fatal result is to be apprehended, but under appropriate treatment recovery is the rule.

Diagnosis.—Heat exhaustion is distinguished from the opposite condition of *thermic fever* by the lowered temperature and feeble pulse. *Syncope* from heart failure or concealed hemorrhage may present identical symptoms, but fortunately the treatment is the same in either case.

Treatment.—If the patient is unconscious, the use of the cold tub-bath, as directed for thermic fever, is not contradicted; in fact, the sharp shock, by restoring tone to the vasomotor system, is of marked and immediate benefit. Following this, and at the beginning in less severe cases, the patient should be placed at rest under a blanket, external heat and friction may be applied, and occasionally moderate stimulation by means of brandy, whiskey, or aromatic spirit of ammonia may be required.

THERMIC FEVER.

(Sunstroke; Insolation; Heatstroke; *Coup de Soleil.*)

Etiology.—Exposure to intense heat, either that of the sun or from furnaces, etc., within doors, is apt to induce high fever, particularly when the atmospheric humidity is so great as to prevent evaporation from the body surface. Alcoholism strongly predisposes to the attack.

Pathology.—Thermic fever is supposed to be due to interference with the heat centre in the medulla, as a result of which more than the normal amount of heat is produced within the body and less is thrown off.

Symptoms.—There may be a few premonitory signs, such as cessation of sweating, dizziness, a sense of oppression, and flashes of color before the eyes. Often the victim falls suddenly and is picked up unconscious. His face is flushed, his skin hot and dry, the pulse is full and bounding, the breathing is labored and sometimes stertorous, and the temperature is high (102°–112°). Twitching, jactitation, rigidity, or even epileptiform convulsions may occur, and in other cases there is active delirium. Frequently the sphincters are relaxed. In unfavorable cases the stupor deepens into coma, the pulse loses its apparent strength, respiration becomes irregular or of Cheyne-Stokes type, and in a few hours the patient dies. In the more fortunate cases the temperature falls, consciousness gradually returns, and the patient recovers in the course of a day or two. Often, however, there persists a certain degree of mental enfeeblement or of inability to withstand exposure to even moderate heat without return of the headache and delirium. This is probably due to a diffuse chronic meningitis set up by the sunstroke.

Diagnosis.—Great care is sometimes necessary in order to distinguish sunstroke from other conditions, notably uremia and cerebral hemorrhage, or to detect the latter when existing. On this account it is essential to examine the heart and the urine in every case which does not react promptly to the bath. A knowledge of the previous health of the patient, gained by interrogation of his friends, may aid in diagnosis. The presence of paralytic symptoms, hemiplegia or spasm, points, of course, to cerebral hemorrhage; but that condition may have antedated the sunstroke.

Prognosis.—This depends upon the severity of the attack, and the promptness with which appropriate treatment is applied. Nearly a third of the cases prove fatal.

Treatment.—Immediately place the patient in a bath-tub filled with cold water, to which ice is freely added, and rub the surface of the body vigorously with the cold water or the ice. In the course of from five to fifteen minutes, or when a thermometer placed in the rectum indicates the approach of the tem-

perature to normal, wrap the patient in a sheet wrung out of ice water, and place him at rest in bed. Administer *aconite*, *belladonna*, *glonoin*, or other remedies according to the indications present. In some cases abdominal pain and diarrhea may be present, calling for such medicines as *arsenic*, *cuprum*, *cuprum ars.*, *podophyllum*, or *veratrum alb.* Uremic and hemiplegic cases should receive the treatment appropriate to those conditions.

DISEASES OF THE MUSCLES.

MYOSITIS.

Etiology.—Inflammation of muscular tissue may be:

1. Primary, probably infective (rare).

2. Secondary to infective or toxemic states, notably gout and rheumatism (so-called "muscular rheumatism").

Exposure to cold air, and especially to draughts, is a common exciting cause. In many cases the condition appears to be due to the presence of some toxic irritant, probably gouty in nature.

PRIMARY MYOSITIS.

Acute simple myositis follows exposure to cold or excessive fatigue. The patient complains of lassitude and pains more or less severe in the affected muscles, and the latter are tender on pressure, and motion is impeded.

Acute infectious myositis may follow the simple variety, but more often its onset is sudden, the patient being seized with a chill followed by fever (101°–104°) and general febrile symptoms. At the same time there is severe pain in the muscles of certain regions, and the latter present a wooden hardness and are exquisitely tender on palpation. The overlying skin is discolored and puffy, and as the result of suppuration of the muscles fluctuation may be detected. A vesicular eruption is sometimes observed. Pyemic symptoms, fever and sweats, are present, and ultimately a typhoid state develops and the patient may die of exhaustion between the fifth and sixteenth days. In more favorable cases the pains abate, the temperature falls, and convalescence becomes established; but the muscular pains and weakness may persist for months.

Acute infectious poliomyositis. After a prodromal period extending over several weeks, during which the patient complains

of lassitude and fatigue, slight edema may be noticed about the ankles or face, and small red macules appear on the face, extremities, or abdomen. At the same time pain develops in the muscles and increases in severity until ultimately the patient fears the slightest movement and so lies stiff and motionless in his bed. The muscles appear to be contracted, holding the limbs in semi-flexion, they do not react to electrical stimulation, and the deep reflexes are abolished. The patient may have a succession of chills, the temperature rises to 104° or more, perspiration is abundant, and a typhoid state supervenes. Involvement of the respiratory muscles may lead to death from suffocation or pneumonia, or it may result simply from exhaustion. Rarely, the disease recedes and recovery follows a prolonged period of convalescence.

Chronic or progressive ossifying myositis. In this rare disease the muscles undergo progressive calcification. The process generally begins in the back of the neck, and consists of the deposit of bony plates, or even ossification of entire muscles, throughout the body.

Treatment.—In simple acute myositis rest and the administration of such remedies as *arnica*, *cimicifuga*, *gelsemium*, *pulsatilla*, or *rhus tox.* usually brings about recovery. Should the myositis merge into the suppurative form, the parts may be wrapped in a moist, weakly antiseptic, dressing, and as soon as pus is detected it must be evacuated and the cavity treated on surgical principles. *Hepar*, *mercurius*, *silica*, or *sulphur* may be given internally, and the patient's strength must be sustained by means of highly nutritious food and alcoholic stimulants.

In acute infectious poliomyositis and in the ossifying form no treatment appears to be of any avail.

SECONDARY MYOSITIS.

Etiology.—Muscular tissue may become inflamed through extension of the process from adjacent lesions (*e. g.*, arthritis) or through the action of toxins, usually bacterial in nature. The

muscular degeneration which accompanies the acute infectious fevers, notably typhoid, is often overshadowed by the symptoms of the primary disease. In syphilis and tuberculosis inflammation of the muscles may constitute an important feature of the disease. A distinct type is, however, afforded by—

Rheumatic myositis (muscular rheumatism, myalgia). This is probably more often due to gouty or other diathetic states than to rheumatism. It is characterized by severe pain in certain muscles, such as the sterno-mastoid (torticollis), the shoulder muscles (omalgia), the dorso-lumbar muscles (lumbago), the intercostal muscles (pleurodynia), etc. The only invariable symptom is pain, but in addition the muscle is sometimes swollen, and in some cases there is a slight rise of temperature (100°). The condition may be acute, lasting but a few days, or it may be chronic and of long duration. The **diagnosis** rests upon the exclusion of neuritis (localized pain and tenderness over large nerve trunks) and neuralgia (less affected by motion or heat, more paroxysmal, etc.).

Treatment.—Place the muscles at rest. Apply dry heat by means of a hot water bag or a warm flat-iron. Massage the muscle daily, and administer such remedies as *arnica*, *cimicifuga*, *mezereum*, and *rhus tox*. Chronic cases may require persistent massage, passive motion, and electricity.

MYOTONIA CONGENITA.

(Thomsen's Disease.)

This is a rare disease, congenital and hereditary, characterized by a painless tonic spasm of the muscles, lasting but a few seconds, which develops whenever voluntary motion is attempted. After motion has begun it proceeds with facility. The muscles themselves are hypertrophied, sensibility and reflexes are normal, and the application of either the galvanic or faradic current induces a normal contraction. The latter develops and relaxes very slowly, however, and during the passage of the galvanic current vermicular, wave-like contractions

pass from the cathode to the anode (the *myotonic reaction* of Erb). The disease is incurable, but does not tend to shorten life.

PROGRESSIVE MUSCULAR DYSTROPHY.

Etiology.—In adition to varieties of muscular atrophy due to disease of the lower neuron, there are several forms of muscular wasting apparently due to primary disease of the muscles themselves. In a majority of cases these conditions are hereditary and represent a congenital tendency to defective development.

Pathology.—The muscular tissues may early show an increase of connective tissue and perhaps some hypertrophy of the muscle fibres, but the latter is ultimately followed by degeneration and atrophy of the fibres, together with a deposit of fat about them.

Symptoms.—The features distinguishing progressive muscular dystrophy from progressive muscular atrophy of spinal origin are:

1. Development in early life.
2. The occurrence of true or false hypertrophy of the muscles.
3. The absence of fibrillary twitchings.
4. The absence of the reaction of degeneration.

Varieties.—1. *Pseudo-hypertrophic paralysis.* This form is characterized by—

(*a*) Weakness in certain muscles, especially those of the calves, and a waddling gait.

(*b*) A peculiar difficulty in rising from the ground, to overcome which the patient places his hands on his knees and "climbs up himself."

(*c*) An apparent increase in the size of the muscles, particularly those of the leg and sometimes of the shoulder.

The condition is due to increase of the connective tissue and fat, associated with atrophy of the muscle fibres. Later, how-

ever, the entire mass becomes wasted, and contractions in the affected muscles lead to deformities (talipes equinus, etc).

2. The "*juvenile form*" of Erb is characterized by—

(*a*) Progressive atrophy and weakness of many muscle groups, especially those of the shoulder, upper arm, gluteal region, and thigh. The forearm and leg muscles are involved late in the course of the disease or not at all.

(*b*) The disease usually begins in the adolescent period, between the 15th and 20th years, and is apt to affect several members of the same family.

3. The "*infantile form*" or Landouzy-Déjérine type differs from that last mentioned only in the involvement of the face muscles. It gives the patient a peculiar, dull expression—the "myopathic facies," in which the lips are separated, the lower one being unduly prominent, and the patient is unable to pout. The other muscle-groups are affected as in the Erb type.

Diagnosis.—The diagnosis rests upon the discovery of obvious muscular weakness and wasting unassociated with the reaction of degeneration or other evidences of disease of the lower neuron.

Prognosis.—The course extends over years, but ultimately involvement of the respiratory apparatus or of the heart, or else an intercurrent disease, leads to a fatal termination.

Treatment.—Carefully selected and graduated exercises for the weakened muscles, together with massage, electricity, and general nutritional measures, should be tried. In the pseudo-hypertrophic variety, *aurum*, *kali iod.*, or *phosphorus* may be prescribed. In other cases *argent. nit.*, *arnica*, *arsenic*, *baryta*, *cuprum*, *gelsemium*, *mercurius*, *physostigma*, *plumbum*, and *sulphur* may be considered.

THE INTOXICATIONS.

ALCOHOLISM.

Symptoms.—The intemperate use of alcohol may manifest its effects in three ways, viz.:

1. *Acute alcoholism* (inebriety, drunkenness). The amount of alcohol required to induce this condition varies greatly, and the manifestations likewise differ in different individuals. As a rule, however, the primary effect is one of excitement, with bright eyes, flushed face, and ungoverned tongue. Then the well-known staggering gait develops, speech gradually becomes incoherent, and the drunken man finally falls into a deep sleep, from which he can be partly aroused only to sink back into his stupor.

2. *Chronic alcoholism.* Persistent intemperance in the use of alcohol ultimately leads to chronic gastritis, hepatic cirrhosis, arterio-sclerosis, and other degenerative changes in the tissues. In the meantime it produces many more or less characteristic symptoms, notably muscular unsteadiness, indicated by tremor of the legs, hands, and tongue, and mental sluggishness with impaired will power, defective judgment and poor memory. Gastric catarrh is evident in the loss of appetite, coated tongue, foul breath, constipation, and morning nausea. Dilatation of the stomach and fatty infiltration of the liver are particularly common in beer-drinkers. Multiple neuritis, meningitis, and insanity are less common sequelæ.

3. *Delirium tremens* (*mania a potu*). As the result of the long continued action of alcohol on the brain, and often apparently as a direct sequence to withdrawal of the customary stimulation, this peculiar variety of delirium develops. At first the patient is sleepless and intensely restless, and then he begins to have visual hallucinations, believing himself pursued

by serpents, rats, and various horrible monsters. His terror is pitiable. Frequently he becomes unmanageable in his efforts to escape his visionary foes, and even suicide is not uncommon. Auditory hallucinations may also be present. The muscles are tremulous, the tongue heavily coated, there is moderate fever (102°–103°), and a weak pulse. The patient may continue in this condition for the greater part of a week, recovery being heralded by his falling asleep. In other cases a fatal termination is brought about by vital failure.

Treatment.—*Acute alcoholism* requires little direct treatment; as a rule it is enough to allow the patient to sleep off his intoxication and then prescribe *capsicum*, *nux vomica* or other indicated remedy. Occasionally it is desirable to empty the stomach, and for this purpose tickling the fauces or compelling the patient to swallow an emetic of mustard and warm water (a heaping teaspoonful to the pint) is effective.

In *chronic alcoholism*, unless delirium tremens is present, it is wise to completely withdraw the intoxicant at once. If necessary, *trional*, gr. xv, or *hyoscin hydrobromate*, gr. $\frac{1}{100}$, may be administered to induce sleep. An abundance of highly nutritious food should be given, tea and coffee may be taken freely, and such medicines as *arsenicum*, *nux vomica*, *strychnine*, or *sulphur* may be prescribed. Unfortunately, attempts at reformation are rarely successful. Only by means of a certain degree of restraint, such as an institution alone affords, can abstinence be secured, and even in cases thus controlled relapse is the rule.

Delirum tremens necessitates control of the patient by attendants and at times by physical means, such as a folded sheet or straps. Forced feeding is advisable, the patient being given, at frequent intervals, small quantities of milk, eggs, and soup. Such remedies as *belladonna*, *hyoscyamus*, and *stramonium* are sometimes sufficient to master the delirum, but in many cases the attendant is justified in giving *hyoscin hydrobromate*, gr. $\frac{1}{60}$, or, if there is no nephritis, *morphia.*, gr. $\frac{1}{4}$, hypodermatically, in order to induce sleep. The depression

following the withdrawal of alcohol may be combatted by the administration of aromatic spirit of ammonia or strychnine.

MORPHINISM.

Symptoms.—The morphine habit usually follows long continued use of that drug, under a physician's orders or otherwise, for the relief of some painful affection. The constant craving for morphine requires increasingly larger doses for its satisfaction, and often the patient goes on periodical morphine sprees. In the meantime a characteristic change comes over the victim's mentality. He is irresolute, lacks self-control, and is untruthful to a degree. As the time for another dose approaches, he becomes restless, mentally depressed, and intensely anxious, with a sense of impending evil. These symptoms are temporarily relieved by the drug, but return and are greatly intensified should the morphia be withdrawn entirely. If the habit is continued, the patient develops neuralgic pains, tremor, paresis, and rarely ataxia. Should he reach an advanced age, he becomes sallow and wizened; but as a rule long before that period he has fallen an easy victim to some intercurrent disease.

Treatment.—It is almost absolutely necessary that the patient be placed in an institution under charge of a separate nurse, for in this way alone can the supply of the drug be cut off. Morphine should be completely withdrawn at once, the resulting vomiting, diarrhœa, and insomnia being treated symptomatically. The patient should be nourished with milk and cream, beef juice, or broths; and aromatic spirit of ammonia or strychnine may be administered to combat the depression.

CHLORALISM.

The habitual use of chloral leads to mental deterioration and weakness, and even to a mild melancholia. Various skin eruptions may develop, or, in their absence, slight exertion or alcoholic stimulation may suffice to produce an intense erythe-

matous redness of the skin. This is probably the result of vasomotor instability. The treatment is similar to that of morphinism.

COCAINISM.

The habitual use of cocain leads to rapid moral deterioration, with volubility of speech, bright eyes and dilated pupils, hallucinations of sight, smell, and hearing, and morbid suspicions or delusions of persecution. Mild convulsive seizures and nystagmus are sometimes noted. The treatment, fortunately more efficacious than in the case of morphinism, consists in the withdrawal of the drug, and the administration of nourishing food, and non-intoxicating stimulants.

PLUMBISM.

(Lead-Poisoning; Saturnism.)

Etiology.—Lead may enter the system by the respiratory passages, the digestive tract, or the skin. It affects principally workers in lead, painters, plumbers, and glaziers. Occasionally poisoning results from the use of hair-dyes, cosmetics, or canned foods.

Symptoms.—The manifestations of lead-poisoning are sometimes rapid in their development, while in other cases they extend over a long period of time. *Colic* is a striking symptom; it consists of severe pain, often paroxysmal, in the region of the umbilicus; the abdomen is not distended, as in cases of flatulent colic, but flat, and the pulse may be markedly slowed. *Muscular cramps*, especially affecting the flexor muscles of the arms and legs, and pains in the region of the joints (*arthralgia saturnina*) are common. *Constipation*, a *blue line* on the gums at the point of contact with the teeth, and anemia attended by a sallow skin (*icterus saturninus*) are usually present.

Muscular paralysis, especially affecting the extensors, and leading in typical cases to wrist-drop, is a serious result of lead-poisoning. The paralysis may affect single muscles or

groups of muscles, it is usually bilateral, is often accompanied by tremor, and may eventuate in rapid muscular atrophy, associated with the reaction of degeneration. Involvement of the central nervous system (*lead encephalopathy*) may produce such conditions as amaurosis, optic neuritis, headache, stupor, delirium, mania, convulsions, or insanity. Gout and interstitial nephritis are also well-known consequences of lead-poisoning.

Diagnosis.—The diagnosis rests upon the history of exposure to lead, the blue line on the gums, and the presence of some of the symptoms detailed above.

Prognosis.—This depends upon the degree to which the system is saturated with lead, and the extent of the changes induced by that poison. Colic and constipation are usually relieved by treatment, arthralgia and palsy are less hopeful, the presence of muscular atrophy constitutes an unfavorable symptom, and encephalopathy and chronic nephritis are rarely cured.

Treatment.—The intense pain of lead colic may be relieved by hot applications or a hot bath, and the use of such remedies as *alumina*, *belladonna*, *nux vomica*, and *platina*. Rarely an opiate is necessary. Constipation may be relieved by the use of *opium*.

The elimination of lead from the system is favored by the persistent use of hot baths, attention to the activity of the bowels, and the use of *sodium iodide*, gr. j-v, taken in half a glassful or more of water before meals. Faradism may be applied to paralysed muscles, and nourishing food aids in combating the anemia. The patient must, of course, change his occupation if permanent recovery is to be secured.

ARSENICAL POISONING.

Etiology.—Arsenical poisoning may be acute, following the ingestion of Paris Green or "Rough on Rats," or it may be chronic, developing in those who occupy apartments decorated with arsenical wall-paper, or in those engaged in handling

artificial flowers or clothing containing the poison. It may follow the long-continued medicinal use of the drug.

Symptoms.—*Acute arsenical poisoning* is attended by intense abdominal pain, vomiting, and diarrhea, and these symptoms may speedily be followed by collapse and death. These choleraic symptoms are frequently associated with albuminuria, skin eruptions, and, if life be prolonged, paralysis from arsenical neuritis.

Chronic arsenical poisoning is attended by symptoms of debility, with anemia, gastro-intestinal irritability, gastralgia, and vague nervous symptoms. Multiple neuritis, with its characteristic "steppage" gait, may ensue.

Treatment.—In cases of acute arsenical poisoning, neutralize some solution of ferric chloride with sodium carbonate or magnesia, collect the precipitate on muslin or filter paper, and administer it freely to the patient. In cases of extreme emergency, dialyzed iron, tincture of ferric chloride, or Monsel's solution may be given. If the stomach has not already emptied itself, administer in addition an emetic of mustard and water, then wash out the stomach by giving copious draughts of warm water, and later administer castor oil in order to carry off the poison from the bowels.

Chronic arsenical poisoning requires discovery and removal of its source, together with symptomatic treatment of the symptoms as they arise.

PTOMAINE POISONING.

(Food-Poisoning; Bromatotoxismus.)

Etiology.—Ptomaines, substances resulting from the bacterial decomposition of organic matter, vary in their toxicity. Decomposed meats, milk, cheese, ice cream, fish, and shell-fish are among the more common substances, the ingestion of which as food is followed by symptoms of acute poisoning.

Symptoms.—At the end of a period ranging from one to forty-eight hours after the ingestion of the contaminated food, the

patient is seized with nausea, vomiting, cramps, and diarrhea. In addition there may be constriction of the throat and difficulty in swallowing, headache, vertigo, dimness of vision, and extreme prostration. Severe cases may closely simulate cholera, the patient perishing in collapse.

In certain cases of poisoning by shell-fish, however, the poison seems to concentrate its attack upon the nervous system. There is intense burning and itching of the face and sometimes the entire body, followed by the appearance of a vesiculopapular eruption; and the patient becomes dyspneic, the face is livid, and delirium, unconsciousness, convulsions, and coma may ensue. At other times the symptoms are simply those of collapse. Death may occur at any time within a week.

Treatment.—If there is any question as to whether the vomiting and purging have cleared the alimentary tract of its contents, a purge may be administered. Stimulants must be given freely, should collapse threaten; and the symptomatic indications are usually present for *arsenicum*, *belladonna*, *bryonia*, *capsicum*, *creosote*, *nux vomica*, or *phosphoric acid*.

ANIMAL PARASITIC DISEASES.

The parasites which infest the body of man are great in numbers and variety, but the majority of them are harmless. In some cases, however, they are directly injurious, and upon their discovery and removal depends the relief of symptoms more or less serious. Of this class of parasites, the various worms are by far the most important, and the commoner forms of the latter are as follows:

1. Cestodes (tape-worms).
2. Trematodes (flukes).
3. Nematodes (round worms, thread worms).

CESTODES.

(Tape-Worms.)

A tape-worm consists of a head (*scolex*) and joints (*proglottides*). Reproduction is by sexual alternation. The bisexual segments breed from the head and the impregnated eggs enter the stomach of some second animal, the *intermediate host*, where the embryo becomes liberated by the digestion of its covering, and then migrates to other organs, where it forms an encysted larva (*cysticercus*). Meat containing these cysticerci is said to be "measly;" should it be eaten without proper cooking by man, the worm develops into maturity in the intestines of the latter. Three varieties are common, viz.:

1. **Taenia solium** (pork tape-worm). This parasite is from six to eight feet long. The head, of pin-head size, is provided with four suckers and a double row of hooklets, and is attached to its body by a threadlike neck about an inch long. Each body-segment contains male and female generative organs, and becomes larger and more mature, the greater its distance from the head.

2. **Taenia saginata** or **mediocanellata** (beef tape-worm) is longer than the taenia solium, the sexual organs in each segment are somewhat different, and the head possesses four suckers but no hooklets. This is the variety most common in the United States.

3. **Bothriocephalus latus** (fish tape-worm) has a club-shaped head provided in the median line with grooves which act as suckers. The segments are short and broad; and the parasite is frequently from twenty-five to thirty feet in length.

Symptoms.—The symptoms arising from the presence of a tape-worm in the alimentary tract are mainly irritative in character, *e. g.*, abdominal pains, nausea, meteorism, and an inordinate appetite. In addition, various nervous symptoms, such as itching of the nose and anus, vertigo, palpitation, headache, choreiform movements, epileptiform convulsions, and marked anemia, may develop. The presence of the parasite is often unsuspected until a segment is passed with the dejecta. Only by the discovery of such a segment or of the ova in the stools can a diagnosis be made.

Rarely the embryos migrate into the tissues of the human body and there become encysted (*cysticerci cellulosæ*). In the brain or cord their presence sometimes gives rise to the localizing indications of a tumor; owing to their development in subcutaneous tissues there may be stiffness on motion; and in the skin they may appear as multiple tumors varying in size from that of a pea to that of a bean.

4. **Taenia echinococcus,** the smallest of the tape-worms, is, when fully matured, about one-fourth of an inch in length and possesses four segments, the first of which, the head, is provided with a rostellum, four suckers, and a double row of hooklets. It infests the larger breeds of dogs. In man it occurs only in the larval state, forming echinococcus cysts. The latter may be found in any portion of the body; the liver is, however, most frequently invaded. *Hydatid cyst of the liver* occasions symptoms which vary according to the size and situation of the growth. A sensation of dragging and pain may be felt in the

hepatic region. Upward displacement of the diaphragm is apt to cause cough and dyspnea; pressure on the portal vein may lead to ascites; pressure upon the bile ducts sometimes occasions jaundice. More significant, however, are the physical signs: fulness in the hepatic region, an increased area of liver dulness, a fluctuating mass or masses, and the hydatid thrill (see page 289).

Diagnosis may be made positive by discovery of the characteristic hooklets in the aspirated or discharged contents of the sac. The case may end fatally in the absence of surgical intervention; following the latter, recovery is usual.

Treatment.—In order to dislodge any variety of these parasites from the intestine, the patient must for two days be allowed only a light diet. On a third day nothing but a little gruel or milk should be permitted, and at the same time the administration of a saline purge is advisable Then, at bedtime, give thirty minims of ethereal extract of *male fern*, and in the morning sixty minims more of the same remedy. A few hours later administer a purge, preferably of castor oil; and watch the stools, in order to be certain, if possible, that the head of the parasite is passed.

Among other effective anthelmintics are infusion of *pomegranate* (two ounces of the bark in a pint of water), *kooso flowers* (half an ounce in half a pint of water) and *pumpkin seeds* (one or two ounces of the powdered seeds mixed with some pleasant substance). The preparatory steps in the treatment must be the same, whichever remedy is used.

TREMATODES.

(Flukes.)

1. **Liver flukes** (*distoma hepaticum*, etc.) are found in the bile passages and the upper intestine. When numerous they are capable of giving rise to cholangitis, hepatic enlargement, diarrhea, and ascites. The ova may be discovered in the stools.

2. **Blood flukes** (*distoma hematobium*) are common only in the tropics. They inhabit the venous system, especially the portal vein and its tributaries, and give rise to diarrhea, ulcerations of the mucous membrane, and hematuria. The ova may be recognized in the urine.

3. **Bronchial flukes** (*distoma pulmonalis*) are met with in China, Japan and Formosa, where they infest the bronchial tubes and cause cough and hemoptysis.

NEMATODES.

1. **Ascaris lumbricoides** (round worm) resembles the ordinary earth worm in appearance, being brownish in color and varying from six to eighteen inches in length. Large numbers of this parasite may be present in the human intestine, and occasionally they migrate into the bile ducts, the stomach, or rarely into the air passages or even the Eustachian tube. As a rule, these worms are harmless, but in some cases they induce symptoms of gastro-intestinal irritation, or, in children, itching of the nose, grinding of the teeth, disturbed sleep, and even convulsions. A satisfactory diagnosis is possible only through the expulsion of one or more worms, but when the presence of the latter is suggested by these vague symptoms the patient should receive such remedies as *cina*, *belladonna*, *cicuta*, *lycopodium*, *mercurius*, *nux vomica*, *podophyllum*, *sabadilla*, *spigelia*, and *sulphur*. Should these remedies prove insufficient, *santonine*, gr. ¼–j, three times a day, or *naphtalin*, gr. iij–xx, t. i. d., followed by a mild purge of calomel, may be given.

2. **Oxyuris vermicularis** (pin worm; thread worm) is a small worm common in and peculiar to man. The female is about half an inch, the male about one-quarter of an inch, in length. They are actively mobile. As long as they remain in the ileum and large intestine they give rise to no symptoms, but should the rectum be invaded, itching of the anus, especially at night, becomes very troublesome, and by invasion of the vagina intense itching and even catarrhal inflammation of that region may be produced. If the worms are not expelled from the

body after the administrasion of *cina*, *mercurius*, *teucrium*, *sulphur*, or other indicated remedy, give *mercurius dulcis* 1x, gr. iii, every two hours until the bowels are moved freely, and follow this by the use of a profuse enema of soap and water, or of plain water followed by two or three ounces of infusion of quassia. Scrupulous cleanliness must be observed, lest by conveyance of the ova to the mouth reinfection should occur.

3. **Trichina spiralis** is a small worm derived from the hog. It enters the human body with imperfectly-cooked food and is set free in the stomach, where it propagates. In the course of a week the living embryos thus brought forth work their way through the intestinal wall into the blood-current and are carried to the muscles, where they assume a spiral form and become encapsulated. Thus they may live for years. During their presence in the alimentary tract the **symptoms** are irritative in character—nausea, vomiting, abdominal pain, and diarrhea, which, in severe cases, suggest cholera. In the course of a week or two muscular symptoms develop, the muscles becoming swollen, tender, and extremely painful. The skin, especially of the eyelids, may be edematous. There is considerable fever (100°–104°), sweating is profuse, albuminuria may be present, and typhoid fever is suggested by the symptoms. The disease is often fatal, about one-third of the cases succumbing to pulmonary complications. **Diagnosis** is usually based upon a knowledge of the infection of a number of individuals by the same meat. Blood examination frequently reveals marked eosinophilia, and inspection under the microscope of a small bit of muscle, excised under cocain-anesthesia, may reveal the presence of the encysted parasite.

Prophylaxis may be secured by thorough inspection of all meats offered for sale, and proper cooking of that which is intended for the table. **Treatment** consists of clearing out the bowels by repeated brisk purges, followed by the administration of glycerine, ℥ss, every hour, or thymol, ʒj in caps. When the trichinæ have invaded the muscles, remedies should be prescribed according to the symptomatic indications present.

4. **Anchylostoma duodenale** (*strongylus duodenalis*) is a short cylindrical worm found in the upper portion of the small intestine. It is rare in the United States, but is quite common in Egypt, Italy, India, and Brazil. It is of great diagnostic importance, because by its constant sucking of blood from the intestinal walls it produces an anemia so profound that it may be classed as pernicious (*Egyptian chlorosis ; St. Gothard's disease*). Gastro-intestinal symptoms are marked : there may be anorexia, nausea, vomiting, abdominal pain, and sometimes obstinate diarrhea. The **diagnosis** rests upon discovery of the eggs in the stools. The disease tends to a fatal issue in the course of weeks or months, but cure can be effected by appropriate **treatment.** The latter consists of the administration of etherial extract of *male fern*, ʒi–ii, or of *thymol*, ʒss every hour for four doses, followed by a purge.

5. **Tricocephalus dispar** (whip-worm) is found in the large intestine in man. It is from one-half an inch to two inches in length, and is called "whip-worm" because of the resemblance of a long spiral thread attached to its head to the lash of a whip. It possesses little diagnostic importance. The treatment should be that advised for ascaris lumbricoides.

6. **Filaria sanguinis hominis** is found principally in the tropics. The mature worm is about three inches in length. The parasite makes its normal habitat in the lymphatic vessels of the human body; its embryos swarm in the patient's peripheral blood vessels during the hours of rest, but disappear during the working hours of the day. The infection of man (*filariasis*) occurs through the agency of the mosquito. Through blocking of the lymph-channels by the adult worm or its ova *chyluria* may be caused (see page 195). The chylous urine may or may not be mixed with blood. The parasites are found in abundance in the sediment. Lymphatic stenosis may also lead to *elephantiasis* of the lower extremity. **Treatment** is usually unavailing, although *methylene blue.*, gr. ij in caps. t. i. d., may be tried, and in the meantime medicines should be prescribed according to the symptomatic indications present.

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